The SURGICAL CLINICS of NORTH AMERICA

Management of Trauma

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J K. Wise

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THE SURGICAL CLINICS

OF NORTH AMERICA

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THE SURGICAL CLINICS

OF NORTH AMERICA

University of Colorado Number

MANAGEMENT OF TRAUMA

WILLIAM R. WADDELL, M.D.
Guest Editor

VOLUME 43, NUMBER 2 • April, 1963

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Recent Symposia

April 1962—*Nationwide*

Cancer Management—Current Knowledge

June 1962—*from Lahey Clinic*

Surgery of the Endocrine Glands

August 1962—*from San Francisco*

Progress in Gynecology and Obstetrics

October 1962—*Nationwide*

Recent Advances in Gastrointestinal Surgery

December 1962—*Nationwide*

Methods of Improving Morbidity and Mortality Rates in Standard Operations

February 1963—*from Chicago*

Clinical Advances in Surgery

Forthcoming Symposia

June 1963—*from Boston*

Complications of Surgery and Their Management

SAMUEL F. MARSHALL, M.D.
CLAUDE E. WELCH, M.D.
Guest Editors

August 1963—*from Mayo Clinic*

Head and Neck Surgery

October 1963—*Nationwide*

Private Clinic Surgery in the U.S.A.

GEISINGER (PA.), HITCHCOCK (N.H.),
JACKSON (WISC.), LOVELACE (N.M.),
PALO ALTO (CALIF.), STRAUB (HAWAII)

December 1963—*from Canada*

Surgery of the Shoulder Region

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W. B. SAUNDERS COMPANY ANNOUNCES $15,000 WRITING FELLOWSHIP GRANT

To mark its 75th Anniversary in 1963, the W. B. Saunders Company, medical and scientific publisher, is making available $15,000 for an unusual medical writing award. A distinguished committee will select the grantee.

The purpose of this grant is to provide financially for a year's leave of absence for an investigator who

(1) Has been doing fruitful and significant biomedical laboratory research over the past several years;

(2) Would like to have time for thought and for preparation of his work in monograph form.

The recipient of the award will not have to agree to publish his monograph with the Saunders Company and will be free to write, instead of a book, a series of journal articles reviewing his research.

Areas of research in the medical sciences and clinical medicine which are acceptable for award consideration are extremely broad, with a preference for those which could be translated into clinical usefulness within the foreseeable future. The investigator should be a resident of one of the Americas, but he may be doing or may have done his laboratory work outside the Western Hemisphere.

Application for the Saunders Writing Award may be submitted in an informal style to the Chairman of the Selection Committee. The investigator should indicate briefly the character of his research and where it has been pursued, along with a short résumé of his scientific background and a bibliography of his important papers.

Members of the committee who will review applications will be men most likely to be knowledgeable about both the applicant and the nature of his work.

(Continued on next page.)

The Selection Committee is as follows:

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Applications should be submitted between *January 1, 1963,* and *May 1, 1963,* directly to Dr. Robert F. Loeb, care of W. B. Saunders Company, West Washington Square, Philadelphia 5, Pa. A decision on the award winner will be reached by August 1, 1963, and the recipient notified. Formal presentation will be made at an award dinner in October, 1963.

Symposium on the Management of Trauma

FOREWORD

THE MATERIAL in this volume was presented by the authors during the 1962 Trauma Course given by the University of Colorado Medical Center and the Colorado Chapter of the American College of Surgeons. The contributions of Dr. Edwin F. Cave and Dr. Preston A. Wade to that undertaking were exceedingly valuable, and their participation is continued herein. We are indeed grateful.

The selection of papers was not made on the basis of any set pattern, but rather in conformity with the editor's appraisal of the interest and value of ideas and data presented. Wherever possible, recent clinical experiences have been analyzed and compared with those reported from other hospitals. Some of the subjects were included because of their importance in civilian practice and the need to fill the gaps between war wounds and the home front assault by high speed vehicles. This applies particularly to the group of papers about abdominal injuries.

It will be apparent that some of the contributions concern unique experiences arising from the geography and climate in which we work. Snake bites and frostbites, as well as high speed highways, contribute to the problems in vascular surgery, to say nothing of the damage inflicted by firearms and ski poles. It is hoped that some of the commonplaces of Colorado will qualify as recondite for others, and that an actual need will be fulfilled by these discussions of our problems and the manner in which they have been met.

William R. Waddell, M.D.

Guest Editor

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The Past and Present of Trauma

EDWIN F. CAVE, M.D.*

Surgery began with trauma. Prehistoric man trephined skulls following injury as well as for the treatment of headache, vertigo and epilepsy. The first trephined skulls were found in France as early as 1685 and they were also discovered in New Guinea and the Bismarck Archipelago during World War II.

The Egyptians in 1500 B.C. treated wounds with "fresh flesh," kept in place by a bandage for a day; later dressings of fat and honey were used; and they treated fractures by splinting. In the Bible, Isaiah speaks of wounds which "have not been closed; neither bound up; neither mollified with ointment." The only surgical operation mentioned in the Great Book was that of circumcision, which was traumatic enough, in that it was performed by the priests, who used a sharpened stone.

The Hindus splinted fractures with bamboo and in Homer's Iliad a wounded soldier is told to "sit where thou art and drink the bright wine 'til Hekamede of the fair tresses shall heat water and wash away the clotted blood."

Hippocrates was a surgeon as well as a great physician (Fig. 1). He set fractures and reduced dislocations, using a special table; he trephined skulls for "wounds of the head"; and his use of tar in the treatment of open wounds was a forerunner of the antiseptic method as we know it today.

Celsus in about 25 A.D. wrote one of the first medical books. In this book he discussed the treatment of wounds and mentioned the four cardinal signs of inflammation: calor, rubor, tumor, dolor; namely, heat, redness, swelling and pain.

Albucasis, who lived in the latter part of the tenth century (936–1013), wrote a book on fractures and described surgical operations as of two kinds: "(1) those which benefit the patient and (2) those which usually kill him."

In medieval medicine, the earliest reference to surgical anesthesia was made when Dioscorides in the first century wrote: "The wine of the bark of the root is to be given to such as shall be cut or cauterized."

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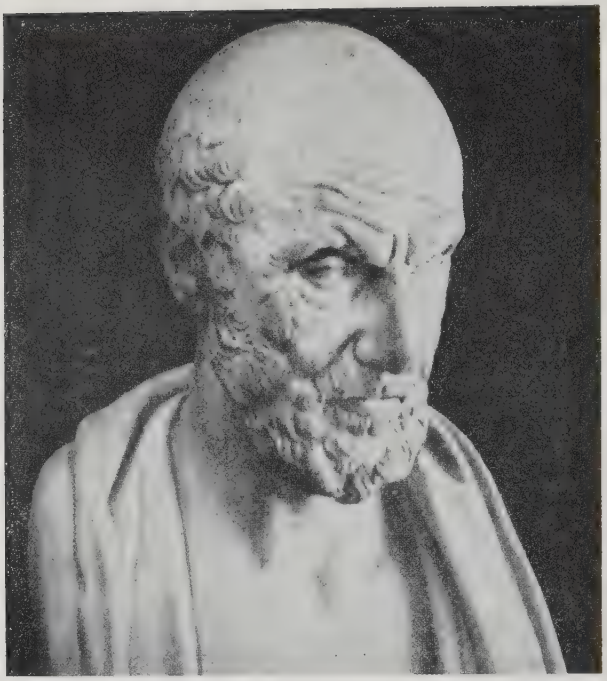

Fig. 1. Hippocrates (460–370 B.C.). (Greek marble bust in the British Museum.)

War advances surgery on all fronts, particularly in the management of trauma. It was gunpowder that drew surgery into the Renaissance movement. The leading surgeons of the fifteenth and sixteenth centuries and of other periods were army surgeons. Ambroise Paré (1510–1590), regarded as the father of modern surgery (Fig. 2), was the greatest surgeon of the Renaissance and one of the greatest of all time. He was surgeon to four French kings and wrote on the life of an army surgeon in the sixteenth century as follows: "I was looking at four wounded soldiers who were propped against a wall, their clothes smelling of gunpowder. An old soldier came up and asked me, 'Sir, is there any way to cure them?' I said No. Whereupon the old soldier went up to them, drew out his sword and cut their throats gently and without illwill." From Paré came the expression, "I dressed the wound and God healed it."

The eighteenth century saw the rise of the English school of surgery. Percivall Pott (1713–1788), a surgeon of St. Bartholomew's Hospital in London (Fig. 3), described the clinical entity of ankle fractures. Pott was referred to as a lively and sociable man and was popular with all ranks of society. He himself sustained a compound fracture of the tibia when thrown from a horse. The limb was saved from amputation, which was readily done for compound fractures at that time. Pott did much to place surgery on a rational basis and bring it into line with the new

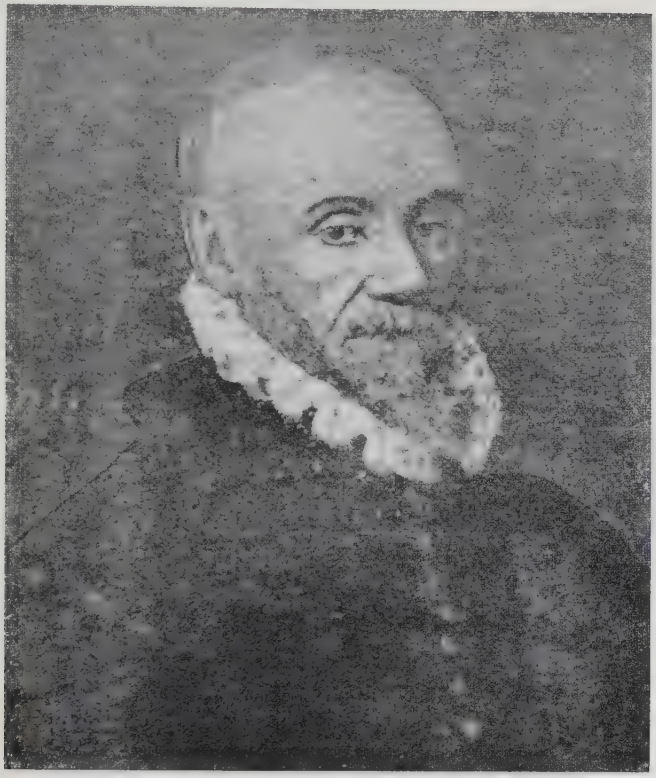

Fig. 2. Ambroise Paré (1510-1590). (An unsigned portrait in the possession of his descendants. La Paulmier)

thoughts of physiology and medicine. John Hunter was his pupil (Fig. 4).

Sir Astley Cooper (1768–1841) is credited with doing the first hip disarticulation (Fig. 5). Cooper had an enormous practice in London and 'tis said his servant, Charles, made 600 pounds a year in tips by showing in patients out of their turn.

In America, Philip Syng Physick (1768–1837), sometimes referred to as the father of American surgery, trained under John Hunter in London and later became surgeon to the University of Pennsylvania. He modified and improved the treatment of fractures.

Two important milestones made possible the modern treatment of fractures and other trauma; namely, the discovery of anesthesia, which was first publicly demonstrated in 1846 (Fig. 6) and, secondly, Röntgen's contribution of the x-ray (Fig. 7), which occurred just 50 years later in 1896. In between came Pasteur's epoch-making investigations in 1857, and Lister's discovery of asepsis and antisepsis in 1867. These great contributions awakened the surgical world. They permitted

Fig. 3. Percivall Pott (1713–1788).

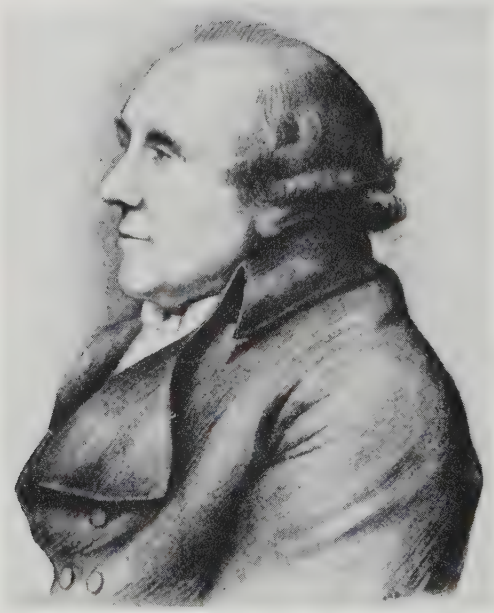

Fig. 4. John Hunter in 1793, aged 65. He died on October 16, 1793. (After the pencil drawing by Nathaniel Dance Holland, Bt.)

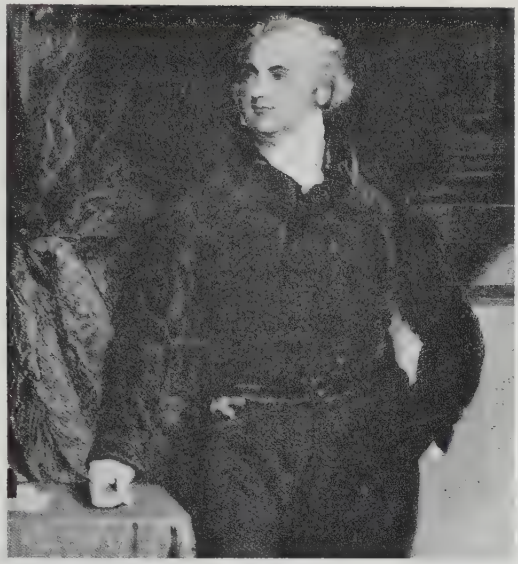

Fig. 5. Sir Astley Paston Cooper (1768–1841).

tremendous advances in the development of the modern treatment of fractures and other forms of trauma.

Pasteur's fundamental observations and Lister's application of them prompted the development of surgery as we know it today; and as it is logical to divide the history of surgery into two epochs: before Lister and after Lister, 1867, so it is also reasonable to divide the history of the treatment of fractures into two periods: that before anesthesia in 1846 and subsequent to that date. The principles in traction and splinting of fractures had, as we have seen, been quite effectively applied since prehistoric times, but adequate manipulation of broken bones and the care of wounds were dependent upon the ability of the physician to render the patient insensitive to pain. Surgeons became skillful in manipulative procedures. Hugh Owen Thomas was the real pioneer in bone setting. Sad though it is to relate, this art is today not applied as universally or as skillfully as it was before the operative treatment of fractures developed. Let us remember that the vast majority of all fractures can still be managed by closed manipulative measures, followed by splinting.

The operative management of fractures had been advocated sporadically, only to be abandoned for many generations, but it was more firmly established early in the century by Sir Arbuthnot Lane of Guy's Hospital (Fig. 8) in London. Lane believed that the "no touch technique" was the major factor in a successful open reduction. His chief difficulty, however, lay in the fact that the plates and screws he employed corroded in tissue, causing reaction, and they frequently had to be removed. In 1912 Sherman (Fig. 9) of Pittsburgh also advocated early operative

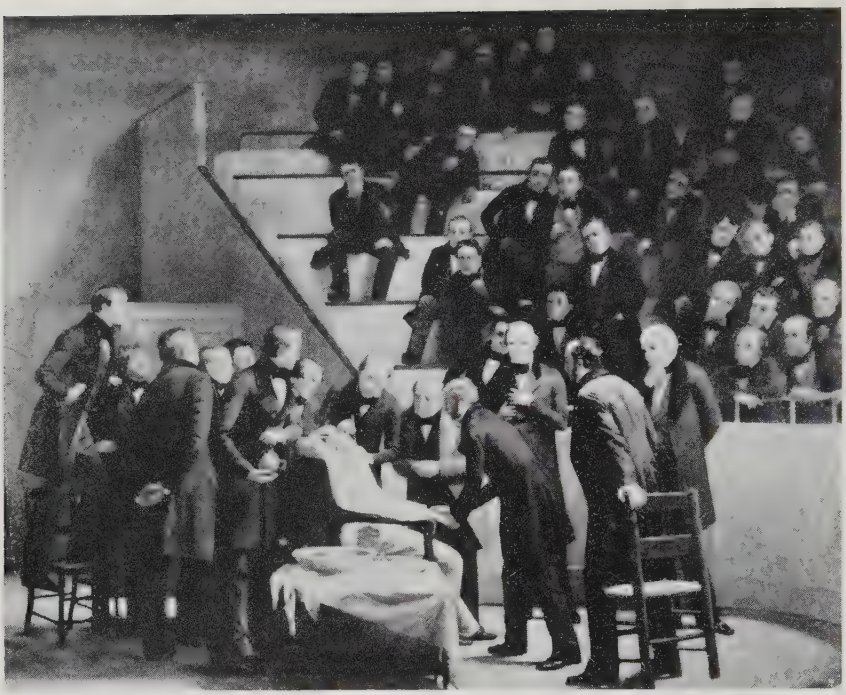

Fig. 6. First public demonstration of anesthesia at Massachusetts General Hospital, October 16, 1846.

Fig. 7. Wilhelm Conrad Röntgen (1845–1923) at the time of his discovery of the "x-rays" (1895).

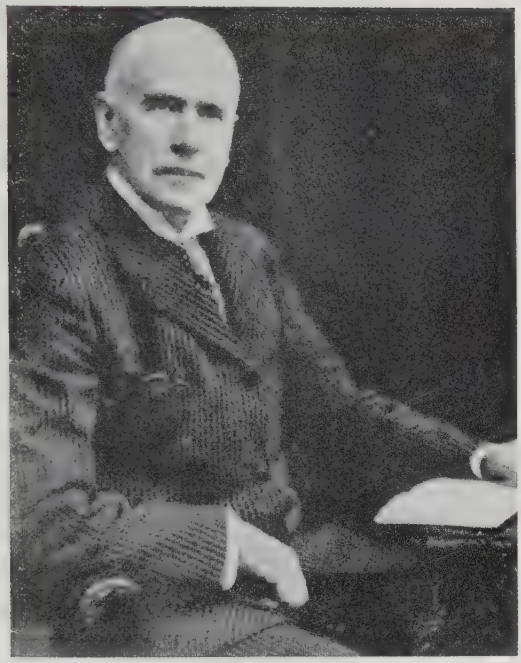

Fig. 8. Sir W. Arbuthnot Lane (1856–1932).

Fig. 9. William O'Neill Sherman (1880–1954).

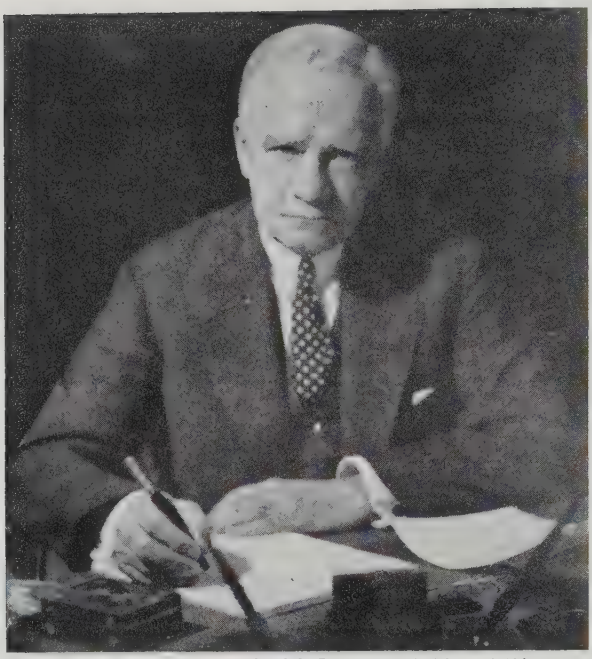

Fig. 10. Marius N. Smith-Petersen (1886–1953).

reduction of long bone fractures. He used Vanadium steel and experienced the same difficulty that Lane had some years earlier. Sherman later abandoned Vanadium steel in favor of 18–8 SMo. stainless steel. In 1929 Venable and Stuck advocated the use of vitallium, an alloy of cobalt, chromium and molybdenum, with small amounts of magnesium, silicon and carbon.

Now the quality of metal is no longer a problem since stainless steel and vitallium are well perfected and are easily tolerated in human tissue if they are properly applied.

The internal fixation of long bone fractures has advanced tremendously and with considerable justification. On the other hand, many abuses of this form of fracture management have come about and many disasters have resulted from the open treatment of fractures. For these disasters we as surgeons must accept the blame. Internal fixation has been applied in the form of plates, screws, wires, nails and bands. The triflanged nail of Smith-Petersen (Fig. 10), first used in 1925, in the beginning was made of a poor quality of metal. Many of the nails disintegrated and did not fulfill the function for which they were inserted. Fortunately, the intramedullary nail came after stainless steel and vitallium had been perfected and certainly the medullary nail has revolutionized the treatment of many long bone fractures, particularly those of the femur, and many ununited fractures of the long bones have been made to heal by

Fig. 11. Ernest W. Hey Groves (1872–1944).

the use of the medullary nail. This form of fracture fixation was used long before Küntscher popularized it during the Second World War: first by Nicolaysen of Norway in 1897, then by Delbet of France, Lambotte of Belgium and Hey Groves (Fig. 11) of England, but in each case the procedure had to be abandoned because of the poor quality of metal. Now medullary nailing is here to stay.

And what of the plastics? Foremost was the pioneering work of Stephen Hudack, who replaced the femoral head and upper shaft of the femur with plastic material in 1946. The Judet brothers of France did an enormous number of femoral head replacements with plastic materials and recorded their experience in the early 1950's. But thus far the body has rejected the plastics, or the structural characteristics of them have not been adequate. The most recent effort is the rigid polyurethane foam, studied and used in a large number of cases by Mandarino of Philadelphia. The name "Ostamer" has been given to this substance. The idea is to inject the substance into the area of the fracture with the hope that it would gradually be invaded by callus and union of bone would follow. Unfortunately, Mandarino's reported results have not been confirmed by other investigators, either in the laboratory or in the clinical application of the use of Ostamer. It is fair to conclude that at the present time no plastic yet investigated has any advantage over the established methods of fracture treatment thus far used; but animal experimentation should be encouraged along this line.

Injuries resulting from war, the high speed automobile, industry, from

sports, and, more recently, from the atomic bomb, have made necessary a wider interest in the overall management of trauma.

The care of the injured patient may be the greatest responsibility of surgery today, and although 75 per cent of all trauma is to the extremities and may well be confined largely to the skeletal structure, the patient with multiple injuries appears more frequently in our accident rooms, and those who survive present dramatic and urgent problems, possibly requiring the services of many surgical specialities. During and after the War of 1939–1945, enormous strides were made in trauma management. Methods of anesthesia, maintenance of blood volume, knowledge of the fluid and electrolyte balances and wound management have all improved greatly during the past 25 years. Recognition of the importance of skin coverage to bone or to soft tissue anywhere following severe skin loss was a most valuable contribution. Perhaps wound management is the most significant part of preventing infection and promoting wound healing, but the number of drugs which have been made available certainly have a place in our armamentarium in the overall management of wounds. Beginning with the sulfonamides in the 1930's, followed by penicillin and, more recently, by drugs with a broader spectrum, they have contributed greatly to wound management and have permitted surgical procedures which were unheard of prior to their manufacture.

It is not within my province to detail the past and present management of specific abdominal, chest or head injuries, but the surgeon who is directing the care of the severely injured patient must have fundamental knowledge sufficient to permit him to evaluate the entire patient and he must first establish priorities of treatment: (1) the airway, either by intubation or, if required, tracheotomy; (2) arrest of hemorrhage; (3) quick splinting of any suspected fracture prior to or after x-rays are taken; (4) accurate appraisal of abdominal, chest or head injury. When these priorities are established the necessary specialists must then be called. In the management of injury the concentration of the various specialties has made for greater knowledge and skill in treating trauma; so much so that the morbidity and mortality from injury are relatively less in the well run accident service.

Open wounds are sometimes easier to evaluate than nonpenetrating trauma, and this is particularly true as regards the abdomen. To quote Sloan, "Current opinion generally takes the point of view that the patient with obscure abdominal injury is better treated by early exploration because the diagnosis can be established and the extent of injury more accurately evaluated. . . . The conservative point of view held 30 years ago is giving way to a more aggressive approach in treating the patients in whom the diagnosis is in doubt."

Trauma cuts across all lines of medicine and surgery and is the commonest cause of death below the age of 36 years. Trauma will always

provide teaching material and it is assuming a more and more important role in all surgical divisions. It will always be with us and may well increase.

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Trauma—The Future

PRESTON A. WADE, M.D.*

I was somewhat shaken by my assignment to discuss the future of trauma treatment, because I do not list prescience among my qualifications as a surgeon. However, the subject began to intrigue me, and I now realize that the Program Committee had more in mind than the mere recitation of one man's prediction. If, during the next few minutes, I can help stimulate your own thinking on the future of trauma management, I shall be satisfied that I have made a modest contribution to one of the most important aspects of this entire symposium.

When we contemplate the tremendous progress that has been made in surgery and in the care of trauma in the past 60 years in this country, we might imagine how appalled the surgeons of the turn of the century might have been had these strides been predicted to them in advance. How could anyone have imagined that a man would be hurled into space, fly three times around the world, and return safely to earth within nine seconds of the time projected for the trip! It would have been equally unbelievable to have been told that some day an extracorporeal machine would take over the function of the heart while this organ was laid bare and stopped in its beat so that certain defects in it could be surgically repaired. How would our former professors have reacted had someone foretold that a kidney could be transplanted from one individual to another, to function normally thereafter! I merely cite these facts to illustrate what we must project in our minds and in our imaginations fully to appreciate what the future in the treatment of trauma may hold.

When I first started by professional career in the early twenties, the major surgical procedures were gastroenterostomy, occasional gastric resection, thyroidectomy and bowel resection. It was still impossible to operate within the chest, and when the first lobectomy was performed, some years later, this was considered a signal feat. Our operations were done then under strange conditions and in great haste. I can remember my professor of surgery telling me that no operation should take more than an hour, and I often assisted him in operations performed in the homes of wealthy New Yorkers who would not entrust themselves to

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hospitals. We transferred equipment, material and personnel to the largest room in the house and proceeded to operate. My professor took off his shirt, donned gloves and a gown, and went to work. As a matter of fact, I never saw Dr. Gibson with his pants off, even in the locker room. He always removed his shirt, hitched up his suspenders, and made no further efforts to formalize operating attire. We used open drop ether anesthesia and, with due respect to our present techniques, I must say we had very few cardiac arrests. As a matter of fact, when a death occurred on the table, we boldly admitted that the patient had had an overdose of the anesthetic agent. These things are laughable in our present state of surgical advancement, so that if I occasionally appear to let my imagination run away with me, I hope you will remember the past.

THE REAL THREAT OF MASS CASUALTIES

To have a proper perspective of what may lie ahead for the surgeon, we must have some idea of the conditions under which it will be necessary for him to act. I do not propose to be a prophet of doom, but I must honestly point out to you that in my opinion, nuclear warfare may be inevitable. Since the day Cain slew Abel, men have been unable to live together without conflict, and throughout the ages, violence, war and sudden injury and death have been a part of man's existence. One has but to walk through an art museum or gallery to see the impact of violence upon our lives. Indeed, works of art inspired by war and violence probably outnumber those depicting pure beauty, love and peace. If we know our history and believe that it repeats itself, we have no other alternative but to accept the fact that, one day, there may be another war, and we must remember that few major wars have been waged without the use of all the possible lethal instruments in the possession of the contending sides. I am not predicting that we will have a war now, or soon, and I fervently hope that we shall never have one, but it seems to me to be quite possible.

We now have nuclear weapons of awesome force that can kill millions of people within a very few minutes of the outbreak of war. We cannot take the stand that if such a war occurs no one would survive, and that it is, therefore, unnecessary to make any preparations, since all will be lost. We cannot accept the premise that nuclear war is so horrible that it will never occur, and that these dreadful nuclear bombs will never be dropped. We know that they have been dropped and, unfortunately, that this country was the first to do so. The fact that modern thermonuclear weapons are more powerful and more deadly than the original atom bomb does not mean that this, in itself, will deter any country from using them. In time of war, each side feels justified in taking any measures

necessary to preserve itself. I recently heard Ambassador Arthur Dean, speaking of the fruitless negotiations with the Russians, tell of the dreadful implications of the nuclear bomb. He described the holocaust that would result if a bomb were dropped on New York City. It would cause a crater 18 miles wide and some half-mile deep, instantly destroy everything in the immediate vicinity and most living creatures for hundreds of miles about, and cause injuries and unknown radiation effects to the rest of the population. These things, as dreadful as they may be, should not deter us from accepting the fact that a conflict may be possible and that nuclear warfare might then be inevitable, and that we must do what we can to prepare for the care of the few that may survive. The world will be different. It will be more sparsely populated. It will be hard on the survivors, but it is still worth trying to preserve.

As one immediate and practical means for those of us who are now here to help in preparing for such a disaster, I should like to call your attention to an effort being made by the Division of Health Mobilization of the United States Public Health Service to educate and direct all citizens in the care of the injured under the circumstances of a nuclear attack. The United States Health Service asked the Cornell University trauma group to draft a manual to be entitled, "For Management of Mass Casualties Under Austere Circumstances," to be prepared under the auspices and with the consultation and help of the Committee on Trauma of the American College of Surgeons. The manuscript is now in the stage of final revision. When this manual has been published it will be distributed through the trauma committees to every possible area throughout the country, for the instruction of paramedical personnel and the laity. I will not go into the details of the contents of this manual except to point out that it is a brief and practical explanation of the simplest care of most injuries when all the usual equipment and personnel are not available. When one realizes that the casualties that may occur in one day from a nuclear attack might number 50 to 60 million citizens, I believe you can appreciate the importance of this work. Obviously, it is of very real concern to all of us.

Most of our hospitals are now being requested to take part in mass casualty drills and to plan programs for the care of large numbers of injured people. Such precautions are necessary not only for wartime emergencies, but as a very important part of every community's effort to handle natural catastrophies demanding careful planning and coordinated effort to save lives and to take proper care of casualties. This work is frustrating and often seems fruitless, but I commend to you the necessity of carrying out these exercises and making the plans, since I believe that this is one of the most important things we can do for the future care of the injured in this country.

ACCIDENT PREVENTION

The past 20 years have seen great progress in the prevention of accidents, and the effectiveness of a concentrated effort is well illustrated in the improvement of the accident rate among industrial workers. At the turn of the century, mishaps among factory and mine workers constituted the major cause of accidental injury and death in this country. As a result of planned accident prevention methods, the industrial worker is now better protected than any other segment of our population. As a matter of fact, there are more injuries in the home and on the highway, and both are increasing while industrial injuries are rapidly decreasing. This improvement has resulted, of course, from a number of factors, not the least of which is the matter of economics. The cost of injury and death to the worker is borne by the employer, and hence the latter's altruism is reinforced by financial considerations. Furthermore, the discipline that can be enforced in an industrial plant is something that is impossible on the highway and in the home. I believe that the future will see great improvements in accident prevention for the populace at large, for children, and for people in public conveyances. It will be very much more difficult to improve the accident rate in the home, but much can be done in this regard by concentrated effort.

Certainly, accident prevention on the highway has great promise. Improved highway engineering will one day make driving much safer than it is now, and automobile travel will be so controlled that every person on the road is not at the mercy of a single reckless or careless individual. It is impossible for us to envision an increase in the number of automobiles in this country without some very definite automated highway control, such as has already been projected. Furthermore, such simple and time-tested devices as the padded dashboard, gear locked doors, dish-shaped steering wheels and, above all, the safety belt, have significantly reduced the incidence of injury and death in an inevitable accident. I am sure that, in the very near future, all cars will be equipped with safety belts and, probably very soon, there will be shoulder harnesses as well. Furthermore, not only will it be unlawful to own a car without proper seat belts, but it will be unlawful to ride in a car without using them. A few years ago a seat belt in an automobile was an oddity, and a person using one was considered somewhat queer. Nowadays, we realize the value of seat belts and it is up to us in the medical profession to help educate the public in their use.

FIRST AID STATIONS OF THE FUTURE

The care of the injured person will probably revolve around those hurt in automobile accidents more than any other victims and it is my opinion

that, in the near and certainly in the distant future, first aid stations will be set up in all parts of this country so that the injured will not be far from adequate help at any place in the country where an accident may occur. These first aid stations will be manned by paramedical personnel, and trained ambulance drivers and attendants will be available at all times to administer first aid and provide careful transportation to a special trauma center or department of a hospital, where proper and more definitive care can be instituted immediately. Today, an injured person may be transported by any one of fifty different types of vehicles, with no standardization of the personnel manning the accident vehicle and no assurance that he will receive proper care even after arriving at a hospital. This seems unbelievable in modern times, and, most certainly, some standards for first aid areas and transportation personnel must be established as soon as possible. In the future, ambulances and other vehicles carrying the accident victim will be carefully supervised so that the haphazard, dangerous, speeding ambulance will be a thing of the past. In my opinion the speeding ambulance kills more people than it saves. Ogden Nash has written a poem which I believe to be appropriate.

> The ambulance travels at a furious rate
> That registers utter defiance of fate
> As dodging through traffic both agile and supple
> It picks up one patient and knocks down a couple.

TRAUMA CENTERS AND THE SPECIALIST IN TRAUMA

I have already mentioned the possibility of trauma centers. The concept behind these is already well established in foreign countries, particularly in Austria and Hungary, where such centers are equipped to receive the injured and to provide care administered by trained personnel experienced in treating trauma of every part of the body. This concept is now being implemented in England and in some parts of the United States. Those of us who have advocated the training of surgeons in trauma have been severely criticized for an attempt to develop a new specialty. It seems quite reasonable, however, that, sometime in the future, surgeons will be trained to take care of injured patients and will make this their life work. While it is true that no one person can learn all there is to know about injury to every part of the body, it must also be admitted that one surgeon can learn a great deal about trauma to every part of the body as well as everything about one single area. This surgeon will be much better equipped to handle a patient with multiple injuries than a collection of specialists in every field. The difficulty seems to arise in determining what special group of surgeons will take the responsibility of developing an interest and experience in trauma. Up

to now, this responsibility has always been carried by the general surgeon, but the orthopedic surgeon, whose interest in fractures naturally makes him responsible for the great majority of injuries, also has a stake in this projected program. I believe it will make little difference which group finally takes on the responsibility, but it is a wonderful opportunity for the orthopedist to develop himself in the care of injury in every field, so that he will one day become the specialist in trauma. He need not know all there is to know about every area in the body. He may not know the intricacies of the removal of a brain tumor, but he most certainly should know how to take care of a middle meningeal hemorrhage. He may not know the technique necessary for removal of lung cancer or open cardiac surgery, but he can very quickly learn how to handle a crushed chest and lung injuries. He will need a training area, of course, and it is my feeling that hospital trauma centers specializing in receiving injured patients will see the need to develop trained men in these fields. Once the heads of surgical departments gain enough insight into this problem to allow the orthopedist to develop in lines other than bones and joints, the training of the young trauma surgeon will be greatly facilitated. In my opinion, this is one of the great challenges to the surgical profession in the treatment of trauma in the future.

STILL MORE CHALLENGES

Problems of Space Travel

The events of the past few months have brought home to us the real possibility of space travel and space exploration. This presents an entirely new set of medical problems for us, particularly in trauma. The possibilities of the need for new concepts in trauma care are so numerous that I cannot begin to list them for you. As an example I could cite one very small problem that is well known to space surgeons. If, while in a room in weightless space, a man is unfortunate enough to pass flatus, the thrust thus produced is enough to hurl him to the ceiling with such force as to fracture his skull! Thus we see that what now is achieved by proper diet and self control to save us social embarrassment, may in the future save our lives!

Transplantation of Organs

When one contemplates the almost inevitable future advances in surgical techniques and surgical treatment of the injured patient, the most important of all would seem to be the possibility of transplantation of organs. At this very moment, we know that bone, blood vessel, and skin can be preserved and used as transplants. We know that spermatozoa can be revived after vitrification and dehydration at low temperatures, and most recently it has been demonstrated that long-term blood

preservation is now a reality. I refer you to the presentation of this subject in the most recent *Journal of Trauma* by Capt. L. L. Haynes and his associates from the United States Naval Hospital at Chelsea, Massachusetts. These men have presented data to show that blood which has been preserved by protected freezing is, as long as five years later, satisfactory for clinical use, and that red cells, resuspended in a serum albumin medium or in their own plasma, can substitute for whole blood even in massive transfusions. The blood, then, is living tissue and is most useful because various elements may be added or subtracted, depending upon the necessities of the case. This is a most exciting development and makes possible the storage of large volumes of blood for mass casualties as well as for daily use. Once the problem of the immunity reaction is solved, and it most certainly will be soon, the transplantation of other organs will become a reality, and there is no reason why we cannot expect that kidneys, and even heart, liver, eyes and lung may be preserved and transplanted when necessary. This now seems a wild dream, but in the future it will be as natural as a simple blood transfusion is today and no more surprising than blood transfusion would have been to our forefathers.

The Dangerous Doctor—An Indictment

Another problem that will test the ingenuity of our research workers will be the control of infection and the problem of the disturbance of the bacterial balance of power by the use of antibiotics. This may be the most important single element in the future of surgery, and, during the process of its development, may raise great problems similar to those we are now experiencing in our hospitals by reason of the resistant bacteria. This difficult problem of infection leads me to remark that in the advancement of all of our programs, we may be creating further problems which we, as physicians, must recognize. In this regard, I would commend for your reading a most instructive book by William Vogt, "The Road to Survival." It is a provocative book and one which is factual as well as somewhat philosophical, but which makes us realize that we, as individuals, bear a large share of the responsibility for the survival of mankind. I should like to quote a passage from a chapter called "The Dangerous Doctor":

"The modern medical profession, still framing its ethics on the dubious statements of an ignorant man who lived more than two thousand years ago –ignorant, that is, in terms of the modern world - continues to believe it has a duty to keep alive as many people as possible. In many parts of the world doctors apply their intelligence to one aspect of man's welfare –survival—and deny their moral right to apply it to the problem as a whole. Through medical care and improved sanitation they are responsible for more millions living more years in increasing misery. Their refusal to consider their responsibility in these matters does not

seem to them to compromise their intellectual integrity. They have been primarily responsible for making Puerto Rico, for example, one of the most miserable areas on the face of the earth, by expanding the population beyond all possible bounds of decent subsistence, and their present efforts to correct the situation are not much more than tokens. They set the stage for disaster; then, like Pilate, they wash their hands of the consequences."

This is a stern indictment of the medical profession and, in some measure, it is very well deserved. I think that in the future we must take our part in anticipating the results of our own work. The obvious, terrible future facing this world with its exploding population is something to which we might well turn our minds. It would be a great deal better if man made plans to control the population at a reasonable level by humanitarian means than to allow the world to become a holocaust of starving individuals. Would it not be unfortunate if the nuclear bomb should, of necessity, become the modern plague to reduce the population to a level where a small percentage may survive? To continue our natural investigation of problems of the future, we must go further in our thinking. This is well presented in another book which I believe every young surgeon should read, "Human Destiny," written by the great scientist, Pierre Le Comte du Nouy. In this book, this renowned scientist who has been responsible for many advances in several areas in medicine, points out that the rapid development of the material side of civilization has aroused the interest of men and kept them in a kind of breathless expectation of the next day's miracle, with little time left for solving the true problem—the human problem. Men have been hypnotized by the incredibly brilliant display of new inventions following one another almost without interruption from 1880 on, and are like children so fascinated by their first view of a three-ring circus that they even forget to eat or drink. He states that the purpose of his book is to examine critically the scientific capital accumulated by man, and to derive therefrom some logical and rational consequence. And he states that we shall see that these consequences lead inevitably to the idea of God. His book, he says, is meant for all who would wish to believe in the reality of human dignity and of man's mission in the universe, for those who do not believe in it yet, and for those who are anxious to be convinced. One cannot contemplate the future of surgery of trauma without having these things come to mind, and it is my hope that, as surgeons, we will not only take our part in the development of our profession, but as du Nouy says, will learn to "believe in the reality of human dignity and consider man's mission in the universe."

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The Healing of Fractures and Nonunion of Bone

EDWIN F. CAVE, M.D.*

After bone is broken, healing occurs in a routine, orderly manner if immobilization of the part is carried out for a sufficiently long time. It is desirable, but not always necessary, to restore alignment and apposition to the main fragments of bone. To produce union of bone after fracture, three phases of cellular activity follow one another: First, the development of provisional callus found in the clot subsequent to the hemorrhage following fracture. Secondly, the formation of primary callus and, third, the union of the fragments by bone. Later, remodeling takes place, resulting from the stresses accompanying normal activity of the individual patient.

THE PROCESS OF FRACTURE HEALING[2]

Within 48 hours after fracture, proliferation of cells destined to form bone begins in the periosteum, the endosteum and the lining layers of the haversian canals adjacent to the fracture (Fig. 1, *A*). Death has occurred in a zone of bone on either side of the break. The proliferation of these cells results in the formation of a cellular wave, which advances from each fragment into the fracture gap (Fig. 1, *B*). The waves of proliferating cells spread across the fracture gap, restoring the cellular continuity of the bone. Cellular differentiation begins almost as soon as proliferation, resulting in the formation of chondroblasts and osteoblasts and of cartilage and bone (Fig. 1, *C*). Finally, the formation of cancellous bone directly by osteoblasts and indirectly by enchondral ossification results in bone union. The zones of necrotic bone are resorbed and replaced by living bone (Fig. 1, *D*).

As fracture surgeons we are interested in what factors are concerned with this normal healing process. First of all, there is the *local blood supply*. Certain areas of the skeleton are anatomically predisposed to

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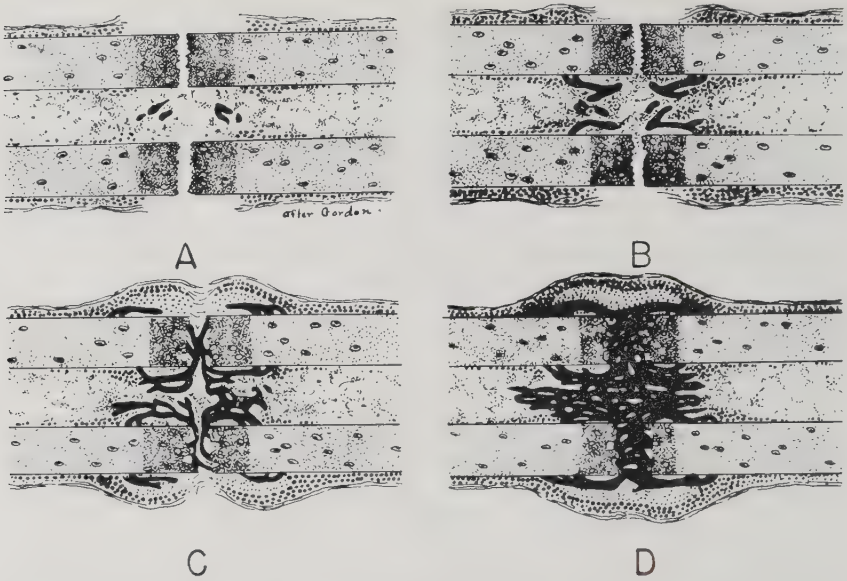

Fig. 1. Normal fracture healing (see text). (Reproduced from Rogers, W. A., in Cave, E. F. [Ed.]: Fractures and Other Injuries. Chicago, Year Book Medical Publishers, Inc., 1958.)

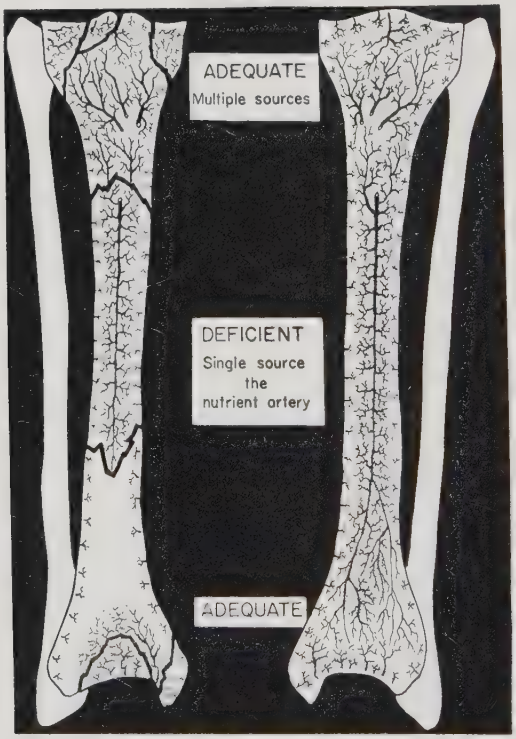

Fig. 2. Blood supply to a long bone. (Reproduced from Rogers, W. A., in Cave, E. F. (Ed.): Fractures and Other Injuries. Chicago, Year Book Medical Publishers, Inc., 1958.)

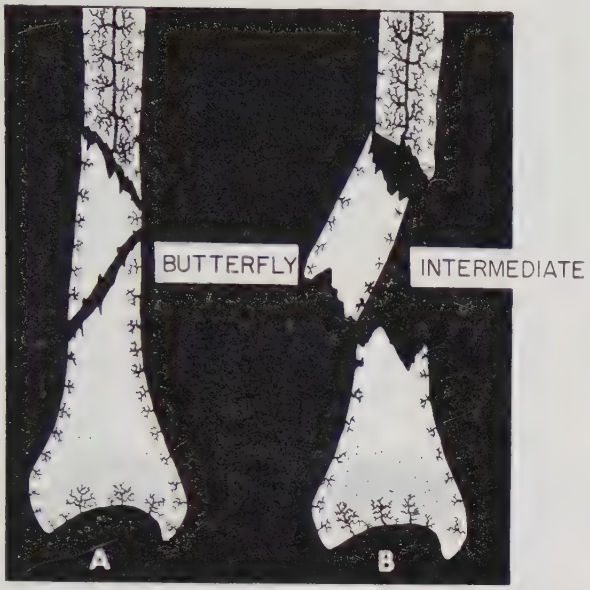

Fig. 3. Effect on healing of (*A*) the butterfly or (*B*) the intermediate fragment (see text). (Reproduced from Rogers, W. A., in Cave, E. F. [Ed.]: Fractures and Other Injuries. Chicago, Year Book Medical Publishers, Inc., 1958.)

delay in healing because the blood supply is relatively deficient (Fig. 2); namely, the femoral neck, the waist of the carpal navicular, the astragalus neck and the lower third of the tibia. On the other hand, certain areas heal readily; namely, the scapula, the pelvis, the ribs, the femoral condyles and the surgical neck of the humerus. In each of these places blood supply is abundant. Generally speaking, the larger the bone the longer the period of immobilization required to promote healing; for instance, the femoral shaft may require four or six months to consolidate, whereas the phalanx of the toe will be stabilized in a few days.

Secondly, *the type of fracture*. In general, the spiral, oblique fracture of the shaft heals more readily than the transverse fracture, presumably because a richer medullary and periosteal blood supply and a more ample source of osteogenetic cells are available in the former. The free butterfly, or the intermediate, fragment, such as occurs in a segmental fracture (Fig. 3), has only its periosteal blood supply, as both the nutrient artery and the metaphysial arteries to the free fragment have been severed.

The third factor is *reduction of the fracture*. Anatomic reduction of a fracture reduces markedly the requirement of callus (Fig. 5). Even if fractures are not completely and accurately realigned, Nature has a way of providing for sound callus formation if absolute immobilization of the part is obtained and is prolonged, unless there has been outside inter-

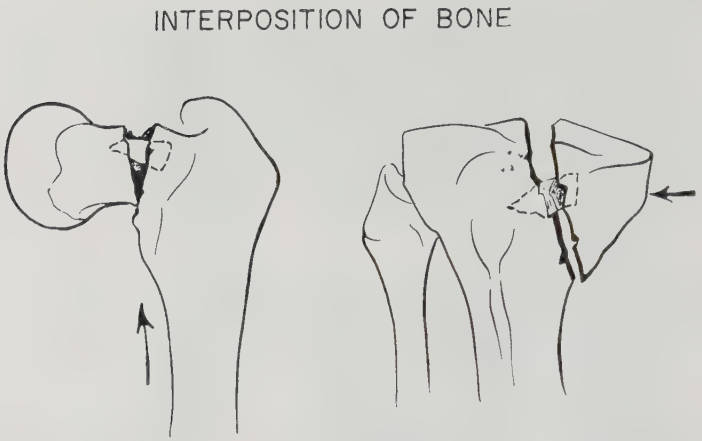

Fig. 4. The interposed fragment at times delays healing. (Reproduced from Rogers, W. A., in Cave, E. F. [Ed.]: Fractures and Other Injuries. Chicago, Year Book Medical Publishers, Inc., 1958.)

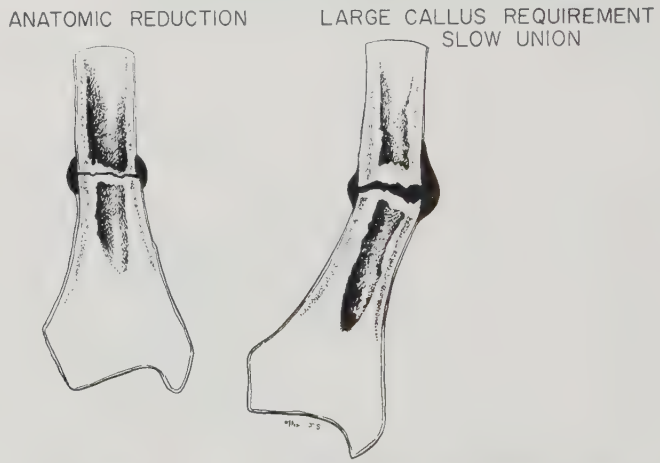

Fig. 5. Callus requirement is increased with malposition. (Reproduced from Rogers, W. A., in Cave, E. F. [Ed.]: Fractures and Other Injuries. Chicago, Year Book Medical Publishers, Inc., 1958.)

ference. This outside interference is usually provided by the surgeon himself.

THE CAUSES OF DELAYED UNION OR NONUNION

It is sad to relate that as knowledge of diagnosis and management of fractures has improved, nonunion of bone is actually on the increase. For this unfortunate state of affairs we must as fracture surgeons hold ourselves largely responsible. Great strides in fracture management have

been made in this century; knowledge of bone healing has improved; techniques of closed reduction of fractures are better; methods of traction, skeletal or skin, are more effectively applied; bandaging material, splints and plaster of Paris are far superior to those used before the Second World War; and metals for internal fixation are relatively inert in body tissue. And yet nonunion is more common because more fractures are being operated upon. Cases are improperly selected. Poor operative technique is being employed—and improper and inadequate postoperative care is prevalent.

In current texts,[1, 3, 4, 5] one may read a long list of causes of delayed union or nonunion of bone (see Table 1), but actually from a practical standpoint there are only three: First, failure of reduction of the fracture; secondly, failure of adequate, uninterrupted immobilization, either by external or internal means; thirdly, the surgeon himself.

In a total number of 160 cases of delayed union and nonunion of long bone fractures we find that only five were classified as delayed union, whereas 155 were classified as frank nonunion of bone (see Tables 2 and 3).

Of a total number of 155 cases, 67 were secondary to open fractures and 88 were closed fractures (see Tables 4 and 5).

As we analyze these cases further and classify the causes of nonunion in the closed fracture we find that improper operative interference was the commonest causative factor.

When we look at the open fractures we find that, next to bone loss,

Table 1. Causes of Nonunion

1. Inadequate reduction	7. Improper application of metal
2. Inadequate external fixation	8. Distraction of fragments
3. Injury to soft parts	9. Too early motion
4. Improper operative interference	10. Too early weight bearing
5. Inadequate internal fixation	11. Sacrifice of bone fragments
6. Too much "hardware"	12. Infection

Table 2. The Location of the Fracture in 155 Cases of Nonunion

	CLOSED FRACTURE	OPEN FRACTURE
Femur	30	8
Tibia	41	53
Clavicle	4	0
Humerus	7	1
Forearm		
Radius	2	2
Ulna	2	0
Radius and ulna	2	3
TOTAL	88	67

Table 3. Causes of Delayed Union in Five Cases of Closed Tibial Fracture

	NO. OF CASES
Improper operative interference	2
Inadequate closed reduction	1
Inadequate external fixation	1
Too early weight bearing	1
TOTAL	5

Table 4. Causes of Nonunion in 67 Cases of Open Fracture

Bone loss	9
Improper operative interference	19
Soft tissue trauma	6
Infection	12
Distraction of fragments	7
Inadequate external fixation	11
Inadequate reduction	3
TOTAL	67

Table 5. Causes of Nonunion in 88 Cases of Closed Fracture

Improper operative interference	38
Inadequate closed reduction	10
Inadequate external fixation	10
Distraction of fragments	11
Too early weight bearing or mobilization	3
Interposed soft tissue	5
Infection	7
Abnormal metabolic bone change	3
Intentional operation to improve motion	1
TOTAL	88

improper operative interference was the most common cause of nonunion of bone.

DIAGNOSIS

Delayed Union

The presence of delayed union must be determined almost entirely by the x-ray film. Certain bones give evidence of healing in more or less prescribed periods of time. The best evidence of bone healing is callus formation as shown by the film. Calcification in periosteum is not true callus formation. As has been stated, the more accurately a fracture is

aligned, the less demand there will be for callus. In such a case, a gradual obliteration of the fracture line without loss of position and the absence of unusual bone atrophy above and below the fracture, or excessive sclerosis at the fracture site, are the best evidences of progressive bone healing. There is no reliable clinical test for delayed union. As a fracture is healing, a manual test for motion at the fracture site should not be made because any movement at the fracture site will have a tendency to interrupt the new bone bridge which is uniting the fracture.

Nonunion

Nonunion of bone occurs when the repair process has stopped completely and it may be suggested by the following *clinical* findings:

1. Pain upon use of the extremity.
2. Progressive bowing of the extremity.
3. Edema of the extremity.
4. Increased surface temperature in the region of the fracture site.

By *roentgenography* the following should be looked for to determine the presence of nonunion:

1. Excess (false) callus production at the fracture site.
2. Gradual bowing or loss of position at the fracture site.
3. Unusual sclerosis at the fracture site.
4. Unusual bone atrophy, below or above the fracture.

PLANNING THE TREATMENT

When the surgeon is confronted with frank nonunion following fracture, he must ask himself the following questions before planning treatment:

1. *Which bone and what part of the bone are involved?*

The trochanteric area and the supracondylar area of the femur will unite more promptly than will the shaft of the femur because the blood supply is more abundant in the former areas. The same applies to the upper and lower ends of the other long bones. The femoral neck and the carpal navicular are notoriously slow in uniting.

2. *How long has nonunion existed?*

The longer the nonunion has existed, the greater the sclerosis of bone ends and atrophy of the shaft beyond the fracture line. Sclerosed bone will require a long time to revascularize even if it is covered by "fresh" bone and nourished by a good muscular covering.

3. *What is the condition of the soft parts?*

Scar in skin, subcutaneous tissue, and muscle will preclude good circulation to the fracture and to the bone graft. The graft must therefore be inserted through an incision over the most viable soft tissue.

4. *What is the condition of the joints above and below the site of fracture?*

Immobile joints will add strain to the fracture site. Therefore, after the graft is applied, the fracture must be protected until there is solid union. Medullary fixation has allowed much earlier joint motion after operations for nonunion.

5. *Is the fracture in satisfactory alignment?*

An ununited fracture in good alignment need not be mobilized by excising scar at the fracture site. Fibrous union gives some stability, and subperiosteal iliac bone may be all that is required to promote union.

6. *To what degree is there sclerosis of bone ends at the fracture site?*

Sclerosed bone, with excessive callus formation, need not necessarily be completely excised; but the shaft should be reshaped by trimming down the excess sclerotic area before applying the graft. Such reshaping will enhance the local circulation and result in easier wound closure.

7. *To what degree is there osteoporosis of bone below and/or above the fracture site?*

Osteoporosis calls for as early use of the extremity as is consistent with healing of the ununited fracture. Until weight is borne or joint motion can be initiated, circulatory exercises may be of benefit.

8. *To what degree is there bone loss?*

Bone loss is, at times, the most severe deterrent to bone healing; it frequently follows gunshot wounds or ill-advised debridement. To promote union, bone must be replaced by grafting or the shaft must be shortened at the fracture site.

9. *To what degree is there shortening of the injured bone?*

Bone lengthening has been largely abandoned as an operative procedure, since it is better to obtain union even though a considerable degree of shortening must be accepted. Shortening of the lower extremity can be compensated for by a lift on the shoe. If the shortening is too great, amputation can be done at the point of election and a prosthesis used if bone union can be established.

10. *If union can be established, will the patient have a useful extremity?*

If, despite bone union, there is severe, painful limitation of joint motion, marked shortening or a useless hand or foot, amputation may be the procedure of choice.

NONUNION OF THE FEMUR AND OF THE HUMERUS

The femur and the humerus can be considered as one category because many anatomical features are common to both and the correction of nonunion can be approached in a similar manner in the case of each bone. One real difference in management is that shortening in the humerus can be accepted if it is necessary to promote union of bone, but is not desirable in the femur.

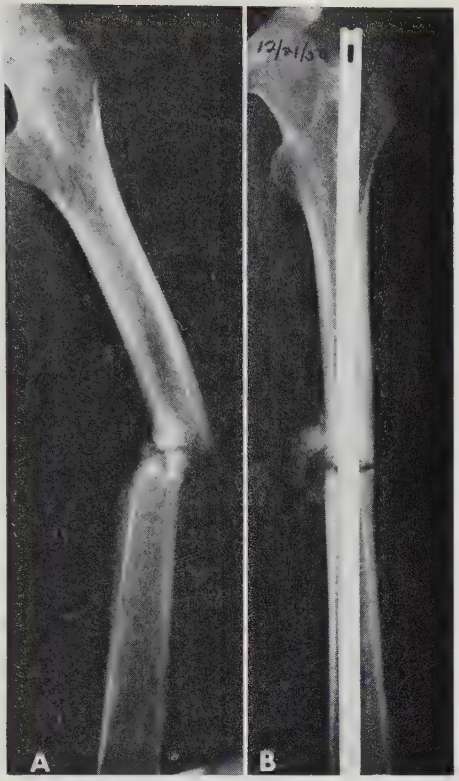

Fig. 6. Delayed union due to manipulative "physical therapy." *A*, Lateral view 6 months after transverse fracture of midfemoral shaft. *B*, Same view 1 month after open reduction and retrograde insertion of clover-leaf medullary nail. Union was progressing. Patient was ambulatory with crutches

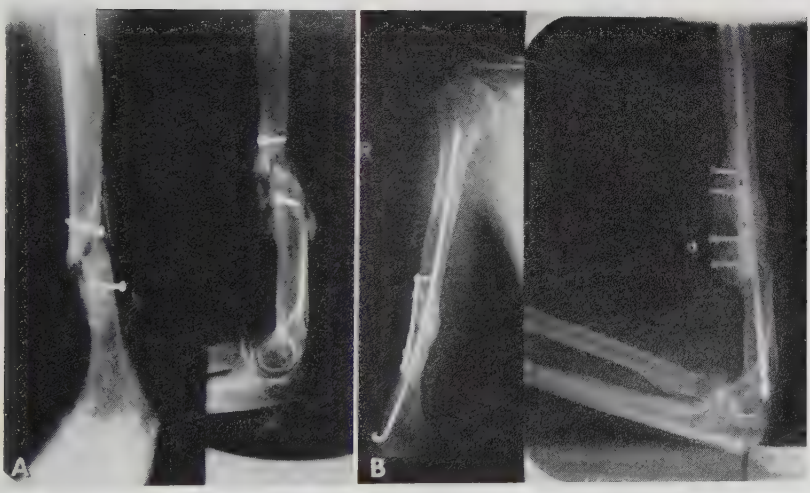

Fig. 7. Nonunion of closed fracture of right humerus, secondary to blow received from a heavy cable. Treated primarily by attempted open reduction followed by attempted bone grafting.

A, Result after application of plate and screws and iliac graft, prior to medullary fixation. *B*, End result 17 months later. Bone union. Elbow motion: 45 degree fixed flexion. Further flexion to 135 degrees. Shoulder motion approximately two-thirds normal. Patient working as a farmer.

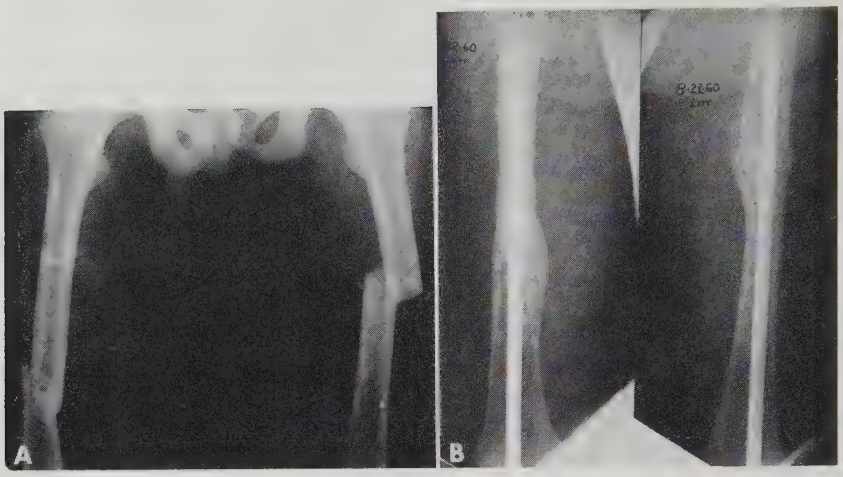

Fig. 8. Nonunion of bilateral femoral shaft fractures secondary to a skiing accident (struck a tree), 5½ months prior to admission. *A*, Original fractures. Primary treatment by skeletal traction followed by plaster spicas. *B*, Treated by 2-stage operation, first on right femur and 3 weeks later on left femur. Intramedullary fixation with bone grafts added. The result was complete union and restoration of normal function of extremities. Patient returned to skiing 2 years after injury.

Since the advent of the medullary nail, the problem of nonunion of the femur and/or the humerus has been largely solved (Figs. 6, 7 and 8). The use of plates and screws for treatment of nonunion of these two bones has been abandoned in many clinics and rightly so. Plates and screws are not trustworthy in immobilizing the humerus or the femur unless there is supplementary external fixation. I cannot be enthusiastic about double plating, although it is advocated by some excellent orthopedic surgeons. When a medullary nail is used, either in the femur or in the humerus, it must provide absolute fixation in all directions. In other words, it must prevent angulation and it must prevent rotation of the distal fragment on the proximal fragment. To meet these requirements the nail must be long enough; it must be large enough in diameter; it must fit firmly in the medullary canal.

NONUNION AND MALUNION OF THE FOREARM AND LOWER LEG BONES

Just as the humerus and the femur are similar insofar as management of nonunion is concerned, so, also, do the bones of the forearm and the tibia and fibula have much in common. The real difference is that in the forearm rotation must be preserved. With treatment of the forearm and tibial fractures (Fig. 9) the medullary nail is of much less importance and many cases of nonunion of the tibia, the radius or the ulna can be managed by the use of plates and screws, supplemented by grafts of

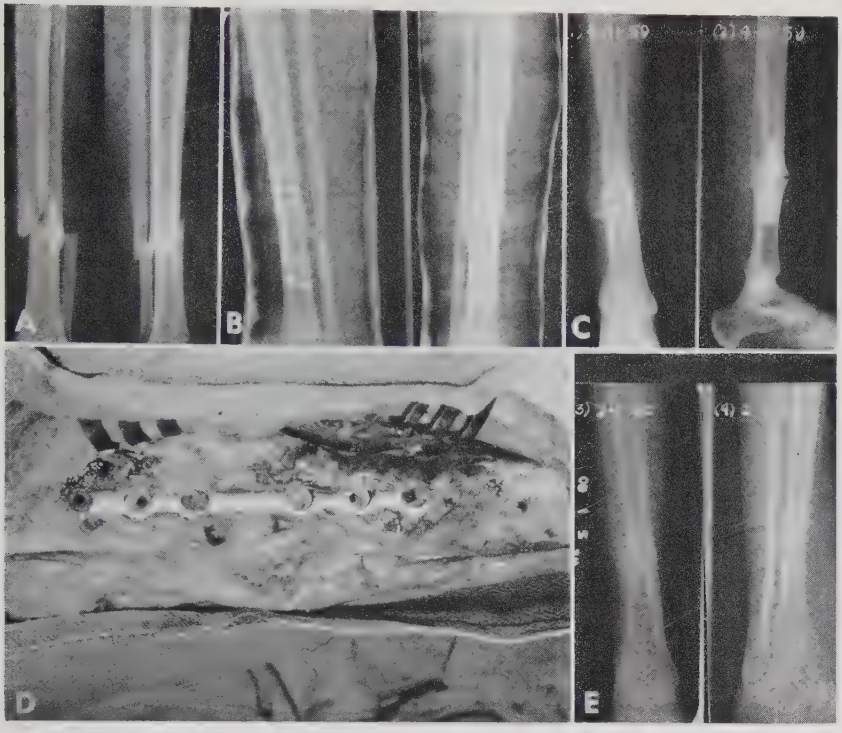

Fig. 9. *A*, Closed fracture of tibial shaft (struck by falling timber). *B*, Treated primarily by open reduction and internal fixation with plate and screws. Ambulation was allowed before fracture was firm. *C*, Nonunion resulted, necessitating removal of hardware, application of barrel-staved iliac bone grafts about the fracture site, followed by plaster fixation. *D*, The appearance of the ununited fracture at the time of removal of hardware. Note avascularization in central portion. *E*, After 26 months. Bone union resulted.

various types, but in practically every instance external fixation must be provided in the form of plaster of Paris.

Nonunion of forearm bones may be handled in various ways. Operative correction is virtually always indicated (Fig. 10). Each bone must be exposed through a separate incision. Internal fixation and the addition of bone are necessary. Internal fixation may be provided by plates and screws, which should always be supplemented by the addition of bone, preferably iliac bone; by a combination of a full thickness graft and screws, the graft to be taken from the crest of the ilium, or from the tibia; or by medullary fixation, supplemented by iliac bone grafts. Practically always external fixation must be provided in the form of plaster.

Malunion of the forearm can be corrected insofar as alignment is concerned, but once the fracture is healed in poor alignment, restoration

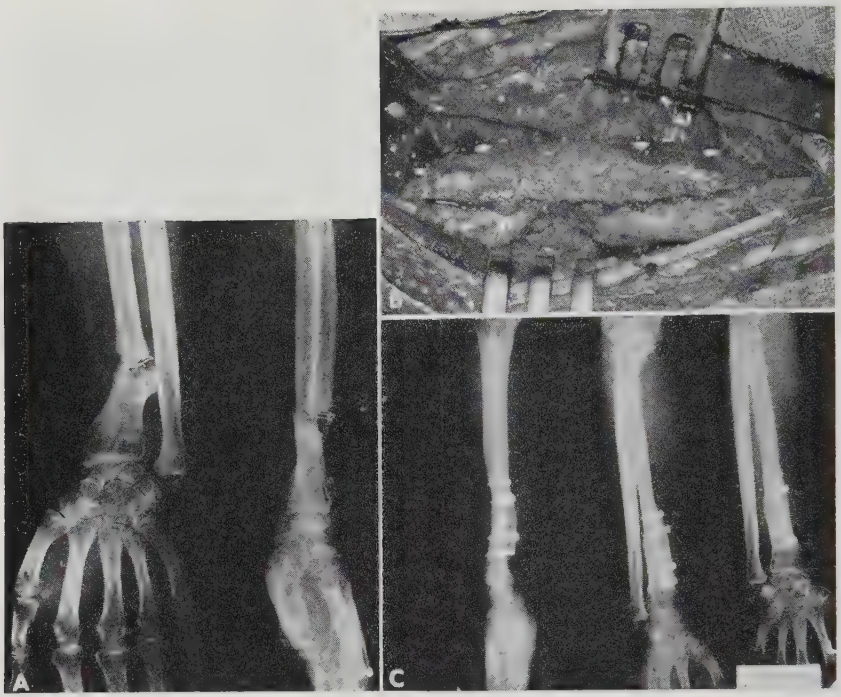

Fig. 10. Nonunion of distal end of radius with bone loss, 13 months, secondary to a closed fracture, due to a direct blow. *A*, Appearance of fracture at time of hospital admission. *B*, Application of iliac bone graft, restoration of length of radius. *C*, Bone union, 3½ months after operation.

Table 6. Number of Operations and Results in 155 Cases of Nonunion

	CLOSED FRACTURE	OPEN FRACTURE
Number of operations		
1	73	50
2	10	6
3	3	2
Multiple	1	0
Failures	0	1
Amputations	1	8
TOTAL	88	67

of pronation and supination is problematical because of the extensive scar contracture of the interosseous membrane. (See Table 6.)

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Spinal Injuries in Sports

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Spinal injuries make up some of the most serious sports injuries, but in total number they constitute only a small percentage. An attempt will be made to clarify how often the spine is injured, and also to describe the types of injuries that occur. An analysis of the mechanism of injury will be presented in order to assist in the prevention and also the recognition or diagnosis of such an injury. Special attention will be given to diagnosis, in view of the fact that the nature of the spine's anatomy makes an accurate diagnosis difficult. Appropriate techniques of physical diagnosis and special x-ray studies are essential in accurate determination of injured parts. The emergency care of the patient with the spinal injury must be understood by all persons concerned with athletic activities.

INCIDENCE OF SPINAL INJURIES

Allan J. Ryan, M.D., of Meriden, Connecticut, Chairman of the A.M.A. Committee on the Medical Aspects of Sports, reports in a recent A.M.A. News that three-fourths of the 25 fatalities in organized football by mid-October, 1961, were related to injuries to the head and neck. R. C. Schneider reports that, of the 14 fatal injuries to the head and spinal cord studied in 1959, four involved cervical spine injuries. It was his impression that there was an overall progressive upward trend in injuries to these areas. In general, it might be said that 50 to 75 per cent of the fatal injuries involve the head and neck, and that of this group, approximately one-fourth involve the cervical spine. In spite of the fact that the cervical spine is vulnerable in all athletic activities, injuries to this area are relatively infrequent. Augustus Thorndike reports in his experience between 1932 and 1954 that there were 46 injuries of the neck and 181 injuries of the lower back among a total of 7394 injuries. These were diagnosed mainly as sprains and contusions. There were 33 fractures of the vertebral processes in this large series of injuries. His

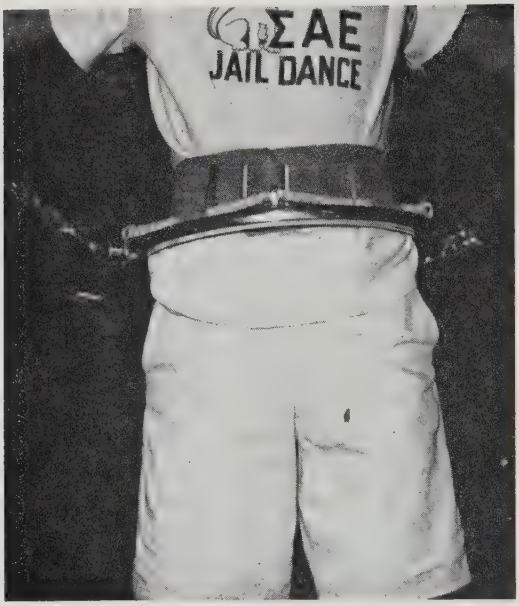

Fig. 1. Serious injuries to the spine occur on the trampoline. Every safety measure should be utilized, including some type of twisting ring. This ring has two control lines and allows rotation in all directions.

experience would indicate that the knee is injured five times to every time the spine is injured.

Injuries to the spine occur in every sport. Ralph Cotton, M.D., formerly of the Air Force Academy, found that 17 of 328 injuries during the spring instructional and intramural sport activities involved the neck and back. Five of the injuries occurred in judo, three in gymnastics, three in rugby, two in diving and one in baseball. During the fall activities, which included football, soccer and lacrosse, there were one-half as many neck and back injuries. It was interesting to note that the average length of time of restricted activities following a back injury was 3.6 days, 3.3 days for a neck injury, as compared to 5.4 days for a knee injury. One would suspect that injuries to the spine would be frequent in wrestling. This was substantiated in the report by Thomas Gonzales that one-half of the deaths which occurred in wrestling involved spinal fractures. The National Ski Patrol report in 1960 showed that 1 per cent of all injuries between 1956 and 1958 involved the spine. During the Winter Olympics in Squaw Valley, Dr. William Stiles found only 11 injuries to the spine in a total of 882 injuries. On the basis of our local experience, it is anticipated that the trampoline is and will be an activity in which neck and spine injuries will occur (see Fig. 1). Experience in the Denver Public Schools has shown that approximately 3.7 per cent of all injuries involved the neck and back. In general, injuries to the spine in all fields of athletics may be considered to comprise about 3 per cent of all injuries.

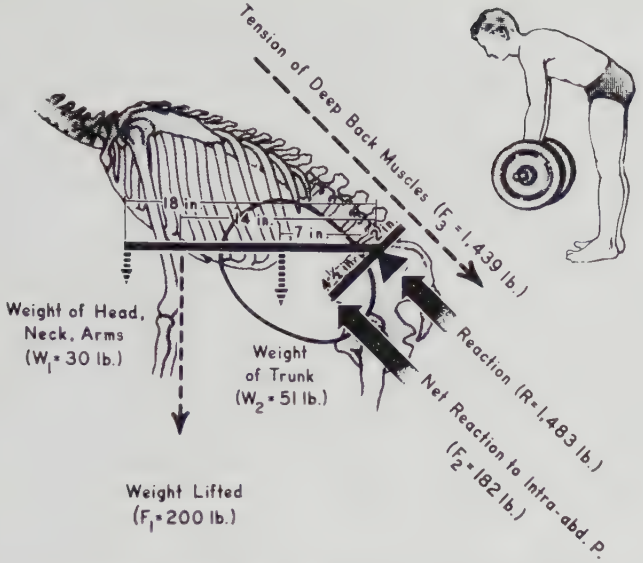
Tension of Deep Back Muscles (F_3 = 1,439 lb.)
18 in.
14 in.
7 in.
Weight of Head, Neck, Arms (W_1 = 30 lb.)
Weight of Trunk (W_2 = 51 lb.)
Reaction (R = 1,483 lb.)
Net Reaction to Intra-abd. P. (F_2 = 182 lb.)
Weight Lifted (F_1 = 200 lb.)

Fig. 2. Counter forces produced by intra-abdominal pressure reduce the force at the lumbosacral joint by as much as one-third. (Morris, J. M., Lucas, D. B. and Bresler, B.: Role of the trunk in stability of the spine. Journal Bone & Joint Surgery *43*: 327–351 [April] 1961.)

The fact that the spine is injured so infrequently in competitive sports is indeed surprising. In contrast, spinal injuries constitute a major portion of industrial injuries. One might speculate as to why the spine is not injured more often. The spine has both intrinsic and extrinsic features which protect it. First of all, it consists of multiple small segments which are relatively flexible, as compared to the lower extremity, which has essentially three mobile joints, with long intervening segments. Anterior-posterior curves in the cervical, dorsal and lumbar portions of the spine also contribute to the elasticity. The vertebral bodies are separated by cartilaginous cushions, namely, the intervertebral disks, which are capable of absorbing energy. Various studies have shown that this shock-absorbing assembly utilizes not only the cartilaginous cushion but also a hydraulic system within the cancellous bone and paravertebral venous sinuses. J. M. Morris has applied engineering techniques to the analysis of trunk support to the spine. The spine is suspended in nearly rigid thoracic and abdominal cylinders, the former filled with air, and the latter with fluid or semifluid materials. When forces are transmitted to the pelvis in lifting, through the spine, and through the semirigid cylinders, calculations show reduction of the forces at the lumbosacral joint by as much as one-third, and in the lower thoracic spine by one-half (see Fig. 2). Reinforcement of the back by the use of thoracic and abdominal counterforces is a phenomenon well demonstrated in wrestlers,

weight-lifters and others engaged in strenuous athletic activities. In contrast, the participant in an automobile accident is usually unprepared for the blow and fails to utilize the extrinsic features of support in the spine to protect himself.

Age may well be a significant factor in the low incidence of spinal injuries in athletes. Anatomical studies of the spine show early degenerative changes in the nucleus pulposus starting in the third decade. Fifty per cent of the patients with herniated intervertebral disks at the Denver Veterans Administration Hospital were between the ages of 30 and 39. Approximately 75 per cent were in the age group of 20 to 39.

Conditioning is also an important factor in the low incidence. The spine and trunk musculature is developed and conditioned in every athletic activity. Although one does not consider the running sports as involving the back musculature, it becomes very evident that the lumbar spine is a major factor in the push-off at the end of the stance phase.

MECHANISMS OF INJURY

The forces that produce injuries of the spine can be divided into compression, flexion, extension, lateral flexion, rotation, and horizontal shear. The effects of compression or compression with flexion consists of a splitting of the vertebral body by the wedgelike nucleus in the first instance, and a compression of the anterior part of the inferior vertebral body in the second instance. Extension force produces essentially the same type of deformity (see Fig. 3). Robert Roaf has shown in specially dissected specimens that rotational shear is extremely important in the production of dislocations and fracture-dislocations. Whereas he was unable to produce dislocations with only flexion or extension, he could readily produce them manually when rotation was combined with flexion or extension. This observation correlates well with the clinical experience that fracture-dislocations, particularly in the cervical area, do not occur in proportion to the forces involved in the trauma. The athlete must be trained to avoid extremes of flexion and extension of the spine, but particularly he must be trained in the techniques to prevent rotation (see Fig. 4). The football player must learn to splint all of the spine with his musculature until he is certain that the play is completed. A hyperextension injury with temporary cord damage occurred in a player with the Denver Broncos this season when he was tackled from behind after relaxing. Fractures of the transverse processes in the lumbar area may result from a direct blow to this area in the same circumstances. With relaxation, the protective splinting of the extrinsic musculature of the spine is removed. Although it is the responsibility of the coach to instruct his players in such protective techniques, by far the best teacher is experience.

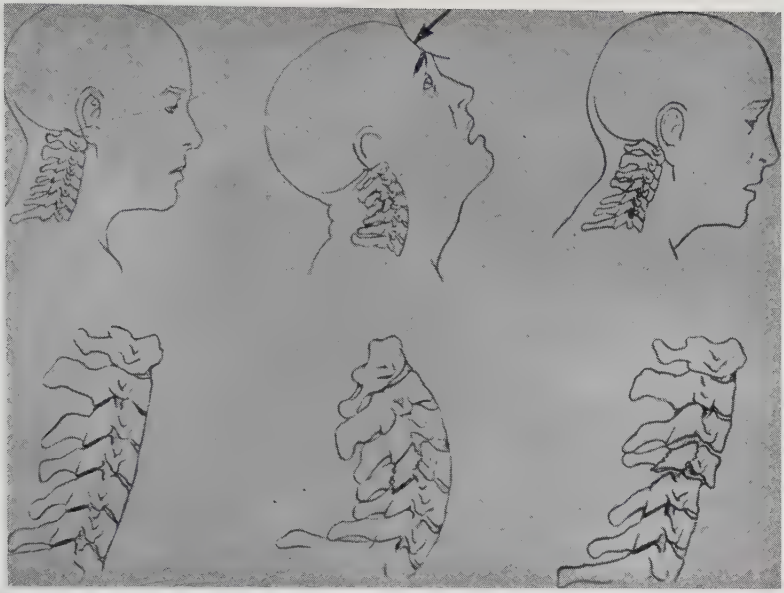

Fig. 3. This type of extension injury produces a radiographic defect suggestive of a flexion injury because of the forward displacement of the superior cervical vertebrae. This deformity results from a fracture through the pedicle rather than a tearing of the interspinous ligaments and capsule. (Forsyth, H. B., Alexander, E., Jr., Davis, C., Jr. and Underdal, R.: Advantages of early spine fusion in the treatment of fracture dislocation of the cervical spine. Journal Bone & Joint Surgery *41-A*: 17–36 [Jan.] 1959.)

Fig. 4. Donald Stone of the Denver Broncos demonstrates how the experienced player protects the cervical spine by avoiding extreme flexion or extension. (Courtesy of Rocky Mountain News, Denver, Colorado.)

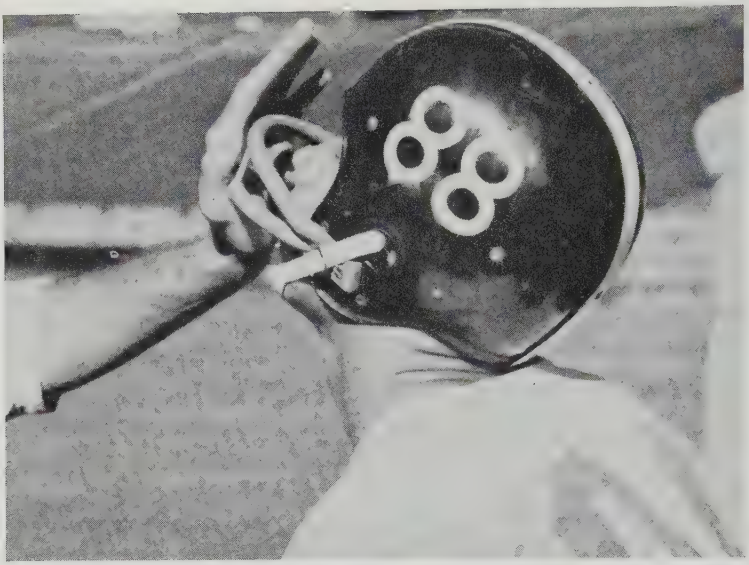

Fig. 5. The face guard, helmet and chin strap, acting as a unit, are capable of increasing the forces on the neck from 30 to 100 per cent.

Schneider et al. have raised the question of the contribution of the face guard to the production of neck injuries. Jim Conley, trainer at the Air Force Academy, noted that during the 1961 season six of 11 spinal injuries involved the cervical spine. He attributes this higher percentage to the face mask. The death of a professional football player in October, 1960 was undoubtedly caused by a hyperextension injury to the cervical spine when the player's face guard was struck by the opponent's knee. The new type of football helmet with its tight chin piece and face guard moves as a unit with the head. The face guard acts as a lever arm, extending 3 to 4 inches beyond the player's chin (see Fig. 5). Simple calculations of the involved lever systems show that, with a distance of 5 inches from the atlanto-occipital joint to the chin, a 100-pound blow on an added 3-inch face guard would be equivalent to a 160-pound blow on the chin. Considering various combinations of face size and face guard sizes from 3 to 4 inches, one can conclude that the forces on the neck are increased from 30 to 100 per cent. Obviously the greatest increases will be in the case of the youngster with a small head and a large helmet. On the basis of these calculations one must conclude that helmets are significant in the causation of neck injuries. It should be emphasized that these lever arms act in a rotational as well as a horizontal plane, and that in the former there is less intrinsic ligamentous and skeletal support, also less extrinsic muscular support. Alterations in helmet and face guard design seem clearly indicated. Schneider has suggested shortening the face mask. In addition, it would seem advisable to alter

Fig. 6. A four-poster collar should be available in every emergency facility for immobilization of any suspected neck injury. This apparatus holds the neck in a neutral position, and prevents extremes of flexion and extension.

the chin strap, making it elastic. It might be possible to design the face guard with a safety release as well.

DIAGNOSIS

The diagnosis of serious fracture-dislocations with immediate pain and loss of neurological function is not difficult. The finding of loss of sensation and motor power indicates serious cord or peripheral nerve damage. Immediate precautions must be taken; as a matter of fact, if there is any suggestion that an injury to the spine has occurred, the patient must be handled as if he has had a serious spinal injury. It is by far safer at this time to "overdiagnose." If there are medical personnel available, they must be called. The patient should be left in the position of injury unless he is apneic and requires immediate artificial respiration or clearing of the airway. If it is suspected that he has had a neck injury, he should be turned carefully on his back by four or five persons working together under medical supervision. One person should maintain the neck in a neutral position with traction. If the traction is maintained, it is extremely unlikely that one could produce more injury. This is true in the presence of a fracture-dislocation. The patient should then be placed on a stretcher, still maintaining the traction. If there will be a delay before transportation is available, traction must be continued. A four poster collar (Fig. 6) is extremely valuable in this instance. This attention and care must be continued not only to the emergency room, but into the x-ray department and until the responsible physician assumes the care of the patient. Fractures or fracture-dislocations of the dorsal and lumbar spine require that the patient be moved cautiously to the stretcher in

Fig. 7. The Boylston views, (*A* and *B*) are valuable in determining unilateral dislocations in the cervical spine. These dislocations are often missed in routine lateral views. (Boylston, B. F.: Oblique x-ray view of the cervical spine. Journal Bone & Joint Surgery *39A:* 1302–1309 [Dec.] 1957.)

either the supine or prone position. It should be stressed that the purposes of emergency care are first to save the injured's life, and second to avoid further damage to the spine and its contents. An effort then is made to transport the patient to a medical facility as soon as possible.

The matter of multiple injuries must be mentioned. A patient with a head, thoracic or abdominal injury may have a potentially dangerous spine injury as well. An unconscious patient must be considered to have multiple injuries until proved otherwise.

Knowledge of the exact mechanism of injury aids in an accurate diagnosis. Information should be obtained from any available witnesses as to the details of the injury. Search for associated abrasions often provides a clue to the mechanism of injury. The telltale forehead laceration indicates an extension lateral tilt type injury to the cervical spine. Local tenderness immediately after the injury is a significant diagnostic sign. If, several hours after an injury, the tenderness becomes diffuse, this sign is of less significance. One can carefully palpate the individual spinous processes. We have learned that much information can also be gained from palpation of the anterior aspect of the cervical spine. As one approaches the lumbar area, palpation becomes more difficult because of the thicker musculature.

Adequate *x-ray examination* is essential. Anteroposterior and lateral views, as well as special views, must be obtained as indicated; a single projection is not adequate. Fracture-dislocations of C-6 on C-7 and C-7 on D-1 are often missed because of inadequate x-rays. In this particular fracture-dislocation, depression of the shoulders is necessary in the lateral views, particularly in individuals with short necks. A special

oblique, cervical thoracic view, with the x-ray tube tilted caudad 10 degrees, will demonstrate the cervical dorsal junction. The oblique flexion views, as described by Boylston, are valuable in demonstrating unilateral dislocation or facet fractures (Fig. 7). Cephalad, oblique anteroposterior views of the lower and midcervical spine, as described by Ruth Jackson, show the facets and lateral masses. Flexion and extension views should be taken under careful control, in order to determine stability of the spine. It is possible to have complete tears of the interspinal or interlaminal ligaments, with instability, which are not demonstrated in the usual lateral x-ray view of the spine. With gentle, controlled motion, increased injury to the spinal cord is not likely to occur. Adequate x-rays of the dorsal and lumbar spine must be obtained as well. However, their techniques present no significant problems. Myelography and discography are indicated only in specific problems.

SPECIAL PROBLEMS

Minor *sprains of the cervical spine* consist of partial tears of the interspinal ligaments, and those of the facet joints. These injuries are not associated with instability of the spine. They respond to rest, heat and massage. Traction is not indicated in the early treatment. Rest, utilizing a felt collar for limited periods of time, is most beneficial. Proper positioning of the neck is essential: it should not be forced into marked hyperextension or out of a protected splinted position. Periods of immobilization should not exceed four weeks, since ligamentous healing in this period of time is sufficient. Removal of the collar and active exercises at the end of this time are followed by rapid improvement. Sprains in the lumbar spine are difficult to treat with complete rest, except in bed. Minor sprains of course can be treated with adequate strapping of the back. The use of a muslin abdominal piece is an excellent technique. This avoids blistering that is often seen with ordinary tape strapping. The firm abdominal pressure also provides considerable relief. The strapping should be applied with the lumbar spine held in flexion.

Fractures of the transverse and spinous processes should be treated symptomatically. Immobilization with a corset for approximately four to six weeks is indicated. It is often true that only fibrous healing of the fracture occurs. However, the associated soft tissue damage warrants adequate rest. At times, associated injuries to the intervertebral disk are more severe than those to the transverse processes. Pain persisting from unhealed fractures of the spinous processes can best be relieved by excision of the fragments.

Compression fractures of the lumbar and dorsal spine, without posterior arch fractures, can be treated symptomatically, with simple corset support. Attempted reduction of the impacted trabecular bone of the verte-

bral body is not indicated. Rarely does the patient have symptoms, in spite of minor anterior wedging. However, should pain persist after a period of six months to a year, fusion of the involved vertebrae only should be done.

Back pain in the teen-age athlete may be due to *epiphysitis* or *osteochondritis of the dorsal and lumbar spine*. The cause and exact nature of this condition is not known. It may not be associated with trauma, may have an insidious onset, and must be differentiated from the injured intervertebral disk. As a rule, the disease is self-limited and responds to decreased activity. X-ray findings may be completely negative. This condition is usually seen after the age of ten years when the secondary centers of ossification appear. Whether it is related to the condition known as juvenile roundback is not known.

The athlete who develops *back pain and sciatica* for the first time has an excellent prognosis. The majority of these patients become asymptomatic with simple bed rest and corset support. In the remaining group, recurrences may result and eventually require excision of a herniated intervertebral disk. When the young adult with disk herniation has failed to respond to nonoperative treatment, the disk should be excised without a posterior arch fusion. This opinion is based on the fact that, in many of these young patients, the greater part of the annulus fibrosus remains intact. Furthermore, the chronic arthritic changes seen in the posterior facets of older persons have not occurred in these patients. Postoperatively, the majority of these patients should be able to return to athletics.

SUMMARY

Injuries to the cervical spine account for approximately one-fourth of fatal head and neck injuries. In the overall picture, however, injuries to the cervical spine and lower back represent only 3 per cent of all athletic injuries. The spine has both architectural features and extrinsic muscular support which prevent injury. Rotational shear appears to be a significant force in the production of fracture-dislocations. The leverage action of the football helmet face guard jeopardizes the cervical spine. Accurate diagnosis is essential in treatment and requires excellent and special radiographic techniques. Treatment of spinal injuries may require modalities of physical therapy and external support, as well as surgical intervention.

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Biomechanics of Ankle Injuries in Skiing as Related to Release Bindings

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Biomechanics is the science dealing with the effect of force and energy upon organisms or organic material. The field of prosthetics has been revolutionized by the application of biomechanical analysis to both upper and lower extremity function. Much of this work has been done at the University of California, through the cooperation of many departments, such as engineering, anatomy and orthopedics. The Crash Injury Research Group, affiliated with Cornell University Medical School, has fostered the study of biomechanics of injuries related to auto and airplane accidents.

Some of the orthopedic-engineering terms that one will encounter in these studies are defined as follows:

1. *Force*—a weight or load applied to a structure is defined by its direction and amount.
2. *Strength* –commonly means a breaking strength; it is the maximum load-carrying capacity of a structure.
3. *Work* –is measured as a force acting through a certain distance in a certain length of time.
4. *Deformation*—bending or displacement of a structure under load.
5. *Elastic bend*—the range in which a material may be bent or returned to its original shape when the force is removed.
6. *Tension*—a force pulling a structure apart.
7. *Compression*—a force bending a structure together.
8. *Torsion*—a rotational force about a longitudinal axis.
9. *Shear*—forces which tend to cause two contiguous parts of a body to slide relatively to each other in a direction parallel to their plane of contact.

What is the magnitude of the forces at play in skiing? At a speed of 30 miles per hour, an average skiing speed, a 150 pound skier develops a momentum (M = mv or momentum = mass × velocity) of 6600 foot-pounds per second; 2250 pound stopping force is required over a distance

of 2 feet. This force increases rapidly in a shorter distance. These forces furthermore, as they affect the musculoskeletal system, are increased significantly due to the leverage effect of a ski on the extremity. Erskine calculates the torque on the tibia in an average ski injury as 600 foot-pounds. It suffices to say that the forces are of such magnitude that they do produce serious injury. This discussion was applied to the ankle because 40 per cent of the ski injuries involve this area. There are two sprains to every fracture of the ankle.

One would like to know what forces the bone will tolerate without producing a fracture. If this figure were known, then a release binding might be designed to be effective at that particular point. Bone is not a simple, evenly formed structure such as a column. Therefore, application of a mathematical analysis is difficult. Furthermore, bone is not homogeneous, but varies in its architecture and in the proportions of compact and cancellous bone. The vascular channels significantly reduce the breaking strength of bone. If one were to compare bone with metals, one could state that bone resembles steel rather than brass. Whereas brass will deform, steel has an elasticity. It is true that bone is brittle and breaks readily. However, bone will return to its original shape after bending if it is not broken. Unfortunately bone has a limited elasticity and, as a consequence, does not absorb energy well, such as would a steel spring. Bone is very much like chalk. Bone resists compression three to four times better than it does tension. As a result, the majority of fractures are the result of tension failures. Shear resistance is good as compared to resistance to tension. Compact bone differs obviously from cancellous bone; the latter has minimal resistance to compression. Whereas compact bone has resistance to compression, it has less resistance to tension.

In considering fractures, tensile strength of bony tissue is of major importance. Most investigators, in testing specimens in the laboratory, have shown the tensile strength to be 9000 to 15,000 pounds per square inch in fresh bone. Male bone averages 30 to 50 per cent stronger than the female bone. It is interesting to note that fibular bone was stronger than that of the femur. Similar samples showed tensile strength of 10,000 pounds per square inch as compared to compression strength of 18,000 pounds per square inch. Average torsional shearing strength was 11,233 pounds per square inch.

Shape of bone affects the strength. Certain structural shapes offer greater resistance to bending or torsion for a given amount of material. In comparing the following structural shapes, a solid rod resists 9 units of bend, an I beam, 53; and a cylinder, 48 units. A cylinder, such as a bone, is superior in resisting varying forces such as torsion, bend and compression. The effect of external supporting structures, such as muscles, has not been calculated for the ankle. A study at the University of

California demonstrated that trunk musculature with its associated intrathoracic and abdominal pressures significantly reduced forces on the lumbosacral spine. Such a mechanical analysis would be necessary to properly evaluate the structural strength at the ankle, including the skeleton and the adjacent muscles and ligaments.

The author measured the cross sections of several specimens of the distal fibula at 1 inch from the malleolus. This determination showed the cross section of the cortical bone to be 0.1 sq. inch approximately. This would indicate that roughly a force of 1000 pounds per square inch would fracture the distal fibula by *tension* alone.

The direction of forces that produce ankle injuries are many. Fractures result from rotation, both internal and external, and from pronation and supination. In an attempt to utilize these findings, manufacturers have produced new release bindings. For example, the Stowe flexible binding features a rotational release not requiring impact, and the Miller binding offers release in many directions, including pronation and supination. Reports regarding these two examples of bindings indicate that when used there have been significant decreases in the number of serious injuries. A study at the American Ski School of Garmish revealed 12 times fewer fractures with the Marker release binding. On the contrary, the American National Ski Patrol report of 1960, which included statistics up to 1958, showed six of ten skiers injured were wearing release bindings.

In summary, an attempt has been made to indicate how a biomechanical approach might be applied to the problem of ski ankle injuries and the design of release bindings. A study should be made of significant forces, the quantity of these forces, energy absorption, and other mechanical aspects. Improved release bindings are available, but improved design will require investigative work. Such a study would be invaluable to those participating in this sport. It might be sponsored by the National Ski Patrol System and the results distributed by this group.

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Index Ray Amputation

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THE INDEX FINGER is the most specialized finger and next to the thumb is the most important digit.[2] This importance is due to its location adjacent to the thumb but separated by a mobile web space. It is always involved in pinching actions, either with or in conjunction with the long finger as a chuck action; it is the finger involved in fine or "fingernail" pinch. It is the most important finger in the stereognosis function of the hand. It provides one of the lateral poles for grasping.

The first dorsal interosseous muscle is a major muscle of the hand and contributes a great deal to the specialized index finger function. It originates from two heads along the adjacent sides of the first and second metacarpal and inserts into the radial aspect of the base of the proximal phalanx (Fig. 2, *A*). In a few cases it may send a few oblique fibers into the hood mechanism but it has little effect on the intrinsic action of the two distal joints.[3] It is innervated by the terminal motor branch of the ulnar nerve. Its action is to deviate the index finger radially and to prevent ulnar deviation in strong lateral pinch. It also provides some stability on the base of the first metacarpal in pinching. In cross section it is two to three times as large as any other interosseous muscle and it is comparably stronger. The second dorsal interosseous also acts to deviate the long finger radially, but its action is much weaker than that of the first dorsal interosseous.

Any appreciable shortening of the index finger (Fig. 1) removes most of its special actions, for which length and dexterity are absolutely necessary. In such a case the long finger takes over pinch and most of the fine functions, but the remaining portion of the index finger hampers some of the actions. The actions of the index finger which are not dependent upon length are dependent upon its being the adjacent digit across a free mobile web space. There is a tale in the Old West about gamblers using the stub of the index finger to deal cards from the bottom of the deck. Adversity has its uses!

There is a myth that normal width of the palm is necessary for strong grasp and using tools and that the laboring man's hand is impaired by any narrowing of the palm. This may be true to a certain degree, but removal of either lateral metacarpal (two or five) does not seem to impair grasp to a significant degree.

Fig. 3. *A*, *B*, Two weeks postoperative. *C*, Note amount of active abduction of the long finger 10 months after the operation.

(Fig. 3, *A*). Figure 3, *B*, illustrates the amount of active abduction of the long finger present with first dorsal interosseous transplant.

SUMMARY

Injuries to the index finger requiring amputation below the distal interphalangeal joint should be considered for index ray amputation, as in many cases the overall function of the hand will be considerably improved by converting the long finger to the highly specialized index finger. This amount of narrowing of the palm has not been a detriment. All patients have been well satisfied with the procedure.

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The Management of Arterial Trauma

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Treatment of arterial injuries has advanced in the past decade to the point where restorative surgery is now feasible for any major arterial injury. In almost all cases, definitive treatment should be initiated to avoid crippling symptoms, loss of limb or organ, or even loss of life. No longer are surgeons justified in ligation of major vessels and serene acceptance of the process of necrosis or viability without function.

Approximately two-thirds of arterial injuries involve the extremities, and these injuries are frequently associated with damage to adjacent bone, nerves and tendons. Injury to the surrounding soft tissue needs careful attention and management according to established principles. The severed nerve is not primarily sutured, the fractured bone can be set later, the tendons can be repaired at any suitable time. The wound is closed primarily only if it has been converted into something similar to an elective wound.

The remaining one-third of arterial injuries are frequently accompanied by trauma to abdominal or thoracic organs, thereby complicating the overall care of the patient.

Favorable reports of treatment of vascular injuries by Jahnke,[5] Hughes[1, 2] and others[3, 4, 6, 9] during the Korean War indicated that remarkable salvage could be attained and gave impetus to the expansion of training in treatment of arterial trauma. Injury to a major artery is not unusual in civilian life. In addition to overt trauma, vascular injuries occur during operations, and physicians in major medical centers daily encounter the problem of potential arterial injury during diagnostic procedures and therapeutic cardiopulmonary bypass.

This appraisal of the experience at the Colorado General Hospital during the past ten years was undertaken to define the extent and scope of the problem of vascular injury in a civilian hospital, to assess current methods of management, and to outline certain principles, techniques and methods which might be helpful to surgical practitioners, particularly those caring for the occasional case.

Fig. 1. Type of hand with an unsightly and nonfunctional short stump suitable for index ray amputation.

The author feels that in an otherwise normal hand an index finger that requires amputation below the distal interphalangeal joint should be considered for a ray amputation, and most cases below the proximal interphalangeal joint would function better with a ray amputation. This usually is not performed as a primary procedure for acute trauma unless it is necessary to amputate through the metacarpophalangeal joint or in the metacarpal. The procedure should be kept in mind and offered to the patient at a later date.

If the index ray near the base of the metacarpal is removed, the long finger then is in a position for specialized functions. This requires a normal web space and a strong radial deviator of the long finger, which can be achieved by transfer of the first dorsal interosseous to the base of the long finger.

Before definitive index ray amputation is performed, a survey should be made to see if portions of the affected finger can be used in the repair, e.g., fillet technique to provide good skin coverage either by direct transfer or the "island flap." (A free mobile web space is, as mentioned above, a necessity and the web space must not be encroached upon during this procedure.) If the first dorsal interosseous is damaged, then if a second sublimis is present it could be rerouted dorsally to the base of the third proximal phalanx to provide abductor power; the removed bone would be available for grafting if needed.

Index ray amputation is not a new procedure[1, 4] but it deserves more emphasis in hand surgery.

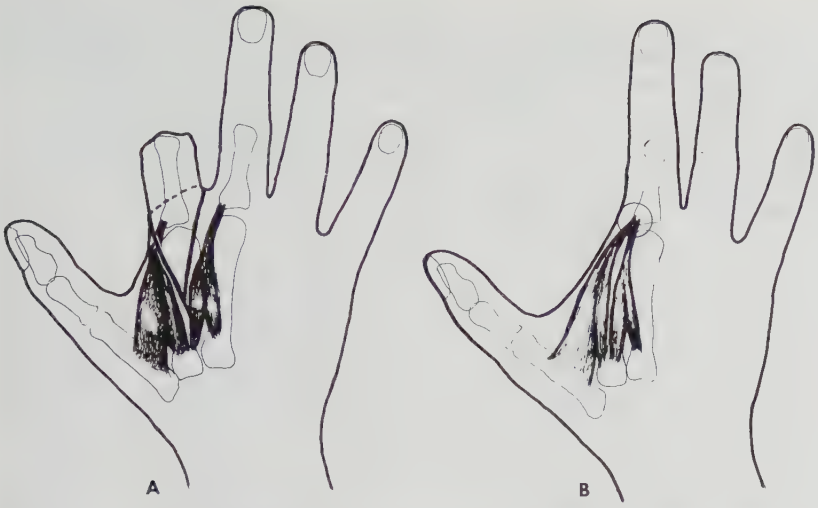

Fig. 2. *A*, Note the anatomy of the first and second dorsal interosseous; also note the outline of the skin incision for index ray amputation. *B*, Transfer of first dorsal interosseous to base of long finger, proximal phalanx.

TECHNIQUE

A tourniquet is used. An incision is made removing a wedge of skin dorsally, circling the finger at the base (Fig. 2, *A*), and then extending through the center of the web space. The first dorsal interosseous tendon is isolated and removed from its bony insertion (Fig. 2, *A*). The second metacarpal is exposed subperiosteally and severed obliquely near its base just distal to the insertion of the extensor carpi radialis longus (a small saw is the best instrument to use). The digital arteries are ligated; the digital nerves are divided cleanly and the ends are placed between the interosseous muscle bodies toward the dorsum. The first dorsal interosseous is transferred into the bony attachment where the second dorsal interosseous attaches to the base of the long finger proximal phalanx (Fig. 2, *B*). (Transverse hood fibers may need to be divided and resutured for ease of exposure.) Simple closure of the skin and subcutaneous tissues is performed, and a dressing is applied to keep the long finger radially deviated. Later taping is applied to splint the finger in radial deviation for three weeks.

The author has used this operation in seven cases with first dorsal interosseous transplant in three cases; these three had definitely better function although all cases were improved by the operation. Two patients were laboring men; when grip testing was performed nine years after surgery in one of these patients, he had equal power in the operated hand. He has had no trouble using tools. The cosmetic appearance is excellent

DIAGNOSIS AND INDICATIONS FOR OPERATION

Diagnosis of an arterial injury is not always by obvious signs. Classically, the ischemic extremity appears pale, cadaverous or mottled, cold to touch, with collapsed superficial veins and absence of pulses. However, the signs may be misleading in the presence of arteriosclerosis, shock, exposure to cold, or arterial spasm. Loss of circulation results in sensory and possibly motor loss within a few hours. Sensory loss may be manifested either by hyperesthesia or by complete loss of sensation in the involved area.

Vascular injuries are usually suspected when there is evidence of bleeding, especially in avulsed wounds. However, soft tissue edema may cause bleeding to cease, or may result in actual arterial occlusion, especially when the edema is encased in a fascial compartment. A pulsatile hematoma may develop into a false aneurysm with arterial continuity and distal pulses. The pulses are usually of less amplitude than those in the opposite extremity. All injuries above the knee or elbow should be explored if a peripheral pulse is diminished or absent, provided the patient is not in shock, and occasionally after a trial of nonsurgical sympathetic denervation has been unsuccessful. When venous connection is present, the continuous thrill and bruit of an arteriovenous fistula can be noted. Definite diagnosis is often made only when debridement of the wound reveals vascular injury.

Aortograms are neither indicated nor warranted for the diagnosis of acute arterial injuries.

The most consistently successful results have been obtained in the treatment of injury to brachial and femoral arteries, because of their good collateral circulation and the relatively simple surgical procedures necessary to restore circulation. Sometimes an injured brachial artery may appear not to require definitive repair; however, a number of patients will later complain of "intermittent claudication" in the forearm when this artery has been obstructed, and frank ischemia and gangrene occasionally demand immediate relief.

Whether arterial spasm alone can cause loss of limb or viability needs further clarification; at present it is believed that there is usually some additional reason for arterial occlusion, and these additional factors are sought unless the patient responds immediately to therapy. Spasm caused by stretching of a vessel may occur in patients with fractures of bones of either upper or lower extremity, especially in children. Hyperextension of an extremity, as in patients in traction, may also produce spasm, with ischemia distal to the area.[8] Evidence of direct trauma to the arterial wall is not the only cause of arterial spasm. An overriding fracture may have permitted prolonged shortening of an artery, as in brachial and femoral arteries, and spasm occurs when the extremity is returned to

normal length after reduction of the fracture. Prolonged arterial spasm can bring about changes in the skin, nerves, muscles and joints of an extremity, and, although it tends to disappear spontaneously, it usually persists long enough to endanger the viability of the soft tissues.

A large, tense hematoma in the limb almost always indicates vascular injury, usually from laceration. If the laceration is in the collateral vessels or some branch of a major channel, a hematoma beneath the deep fascia may compromise the flow through the major vessel; this is commonly observed in puncture wounds and at fracture sites, especially in the forearm and in the popliteal space. A perivascular hematoma may dissect from the vessel wall and cause compression of the lumen. Accompanying arterial spasm may result in either complete occlusion or arterial thrombosis. If an intimal tear underlies this hematoma, occlusion from thrombus will result.

TREATMENT

The conservative or nonoperative approach to vascular injuries has been abandoned in favor of an aggressive and confident application of current surgical procedures. The following protocol is not intended to suggest that the techniques and concepts are original, but to indicate methods that have given satisfactory results in this clinic.

First Aid

A tourniquet is seldom necessary and should be used only when pressure directly to the wound area fails to control bleeding. If a tourniquet is applied, it should not be loosened until a pressure dressing has been applied and blood or plasma expanders are available to treat or prevent shock. If hemorrhage is excessive when the tourniquet is released, bleeding may be controlled by pressure proximal to the site of injury. Clamping a vessel blindly in a bleeding wound is to be discouraged, since this may lead to damage of veins and nerves, and may necessitate subsequent grafting of the artery. Sterile technique should be utilized to prevent infection.

Preoperative Measures

First aid is continued and the following are instituted:

1. If indicated, transfusion or plasma expanders are begun, as well as other measures to prevent or aid recovery from shock. Definitive vascular surgery should not be carried out on hypotensive patients, but occasionally control of hemorrhage and revascularization of tissue is prerequisite for control of shock. Under such circumstances, operation should not be delayed.

2. Antibiotics, preferably penicillin and streptomycin, are started in heavy doses.

3. Tetanus antitoxin or toxoid is administered.

4. If the patient is normovolemic following shock therapy, spinal or brachial block anesthesia is advised according to the location of the injury. However, if shock is impending, general anesthesia is preferred. On rare occasions, intra-arterial sympatholytic drugs (Priscoline 25 mg. or Hydergine 0.6 mg.) or sympathetic blocks are used in an attempt to obtain return of pulsations in a vessel injured without laceration; if pulsations do not return immediately, operation is undertaken. Sympatholytic drugs and sympathetic blocks are contraindicated when there is arterial laceration or shock.

Operation

Special equipment must be at hand to be prepared for vascular procedures. The kit should include:

Vascular clamps (Glover, DeBakey, Beck, Gross, Potts, etc.) (If no vascular clamps are available, oiled umbilical tapes are used.)
Braided 5-0 silk on an atraumatic tapered-point needle
Mineral oil
Plastic surgery needle-holder
Fine-toothed thumb forceps
Fine dissecting scissors
Spools of No. 22 and No. 24 steel wire
Ureteral catheter
Heparin, 100 units per kilogram of body weight
Papaverine hydrochloride, 2.5 per cent solution
Autogenous saphenous vein, arterial homograft, or cloth prostheses (Teflon, nylon or Dacron)

After irrigation and antiseptic preparation of the wound, the incision is enlarged over the involved area, the wound is debrided of all nonviable tissue, and hematomas are evacuated. The artery and vein are mobilized above and below the immediate site of injury. This matter of proximal and distal control is fundamental to the successful operative management of arterial injuries. Bleeding is controlled by serrated bulldog or vascular clamps, or by oiled umbilical tapes placed somewhat away from the ends of the vessel, with care to control the collateral as well. Noncrushing vascular clamps are preferable.

Arterial spasm, if present, is controlled by the use of warm saline or by overexpansion of the spastic segment by means of injection of saline under pressure,[7] or, if laceration is present, by overexpansion by means of a blunt-end hemostat. Other methods are injection of procaine around the area and into the sheath of the median or sciatic nerve when exposed nearby, or application of a sponge saturated with 2.5 per cent papaverine

hydrochloride solution for 15 to 30 minutes. The overexpansion method is preferred. If there is no laceration and the spasm does not subside, the involved segment is excised and a graft is inserted or the defect is reapproximated. Likewise a vessel containing hematoma of the perivascular sheath is excised and grafted or reanastomosed, since there is frequently an intimal tear and/or an intraluminal thrombus.

The distal vessel should be investigated for retained clot, and if such a clot is found the vessel is cleared by irrigation from below with a No. 6 ureteral catheter, or the clot is extracted mechanically. The author uses a bent No. 24 wire.

The traumatized vessel is carefully debrided, excising at least 1 cm. beyond the gross injury after laceration and at least 3 cm. after a gunshot wound. After debridement of the ends of the vessel, all loose areolar tissue near the cut end where sutures are to be placed is excised; the adventitia is not removed. Transverse or lateral repair of partially severed vessels is avoided to obviate thrombosis, necrosis with hemorrhage, aneurysm, or narrowing of the vessel lumen with resultant occlusion. In most instances, the vessel should be completely transected and a primary anastomosis done.

Primary anastomosis of the vessel is desired, but if there is the slightest possibility of tension a graft is inserted for restoration of vascular continuity. Conversely, since a vessel elongates and dilates with return of pressure, a graft may result in "buckling" if the length is not properly estimated. Vein grafts (saphenous vein) are used only for the extremities, particularly at joint areas. The vein should be smaller than the artery and reversed because of the valves. Collateral vessels are observed; they are sacrificed only when this is necessary for mobilization.

If the accompanying vein is lacerated, it is ligated unless there is no venous return because of extensive cavitational trauma, in which case preservation of the vein is required.

Single interrupted 5-0 arterial silk sutures 1 mm. apart and 1 to 2 mm. from the edge are used for most anastomoses and closures. The posterior row is performed from the inside (with knots outside) to avoid turning the vessel over. Occasionally a continuous suture is used on the posterior part of an anastomosis, but only on the larger trunk vessels. The vessel wall is not purposely everted with mattress sutures, but care is taken to obtain intima-to-intima approximation.

Before completion of an anastomosis the proximal clamp is removed to "blow out" any retained clot and then reapplied; this procedure is repeated for the distal clamp. The distal vessel is checked for any retained clot by passing a No. 24 bent wire distally just before the final sutures are tied. After completion of the anastomosis, the distal clamp is removed first, allowing time for suture holes to seal with a fibrin clot.

Intravenous heparin, 100 units (1 mg.) per kilogram of body weight,

is given just before the definitive arterial repair is performed. Protamine sulfate or polybrene has seldom been necessary to counteract the heparin effect after the arterial repair is completed. No subsequent heparinization is required.

After restoration of arterial blood to the ischemic part, debridement of the wound is continued, if necessary, and a piece of nonviable tissue is sent for culture. The artery is covered with surrounding healthy tissue, since viability of the graft and prevention of infection at the anastomotic site depend on careful coverage of anastomotic lines and grafts. The skin and subcutaneous tissues may be left open for secondary closure five to seven days later. The fascia should not be closed if there is any question of muscle ischemia. Compartment fasciotomy in forearm (anterior and posterior) or leg (anterior, posterior and lateral) is frequently advisable, especially in delayed or prolonged procedures. Fasciotomies are done through relatively small skin incisions, cutting the fascia widely with scissors.

Both the surgeon and the anesthesiologist should attempt to keep the blood pressure normal by vigorous measures to counteract shock, using blood liberally.

Postoperative Management

If color is poor postoperatively, sympathetic blocks may be performed, or proximal intra-arterial sympathetic drugs may be given. Sympathetic denervation should not be the principal measure to obtain return of adequate circulation. Ganglionectomy is not advised in conjunction with definitive arterial repair. If the extremity is white and cadaverous one hour after operation, if an adequate distal pulse has not returned, or the pulse has decreased in amplitude within six hours, the wound should be re-explored.

Ambulation is begun whenever the patient's condition warrants, usually on the fourth day if the lower extremity has been repaired. The hazards of anticoagulant therapy for prevention of arterial re-occlusion outweigh benefits. However, anticoagulants may be required for venous thrombosis or embolism.

Delayed swelling resulting from muscle ischemia, manifested by tenderness and swelling of the extremity distal to the anastomosis, occurs most frequently in the anterior tibial compartment in the leg and the flexor compartment of the forearm. Fasciotomy of the involved compartments should be done immediately.

Antibiotics should be continued. Infection, hemorrhage and re-occlusion are the most frequent complications. Dehiscence, hemorrhage or thrombosis may occur from inadequate arterial debridement, poor anastomosis, inadequate arterial coverage, excessive tension, unrecognized distal thrombosis, or infection.

MATERIAL

Seventy patients with arterial injuries were treated at the University of Colorado Medical Center during the period July 1, 1952, to September 1, 1962. These patients ranged from 4 to 57 years of age, with the highest incidence between 10 and 25 years. Nine of the patients were less than 14 years old. There were 60 males and 10 females.

Fifty of these patients were injured in accidents outside the hospital. The other 20 were treated for vascular injuries caused by iatrogenic trauma occurring during diagnostic or therapeutic procedures (Table 1). Eight of the 70 patients had no skin penetration.

Table 1. Etiology of 70 Cases of Arterial Injury

	PENETRATING	NON-PENETRATING
Gunshot	18	—
Knife	9	—
Automobile accident	5	5
Glass door	5	—
Fall	3	1
Ski cable	1	—
Ski pole	1	—
Oil barrel	—	1
Industrial wringer	—	1
Iatrogenic trauma (20)		
Therapeutic (12)		
Laminectomy	4	—
Tourniquet (snake bite)	2	—
Herniorrhaphy	1	—
Cholecystectomy	1	—
Retroperitoneal node dissection	1	—
Varicose vein excision	1	—
Osteotomy	1	—
Heart pump	1	—
Diagnostic (8)		
Arterial catheter	5	—
Arteriogram	2	—
Trochar	1	—
TOTALS	62	8

Thirty-four patients had multiple injuries. Twenty-three had fractures or dislocations at or near the site of vascular injury. Nine fractures were compound, and nine patients had multiple fractures. This group of multiple injuries included nine nerve injuries, five bowel perforations, four perforations of the inferior vena cava, three multiple tendon lacerations, and two severe head injuries. Most of the patients had major soft tissue damage in the injured area. Although accompanying veins were frequently injured, they are not listed except for the inferior vena cava.

Thirty-two of these 70 patients suffered shock of varying severity. Sixty patients were treated as emergencies within 12 hours, most of them within four hours. The remaining ten patients were treated 24 hours to nine months after injury, because of progressing or continuing symptoms. This group included two false aneurysms and three arteriovenous fistulas operated from two days to one month after injury. Two of these fistulas were operative injuries which occurred during lumbar laminectomy.

Brachial, axillary, femoral and popliteal arteries were the most commonly injured vessels, and were involved in 50 patients (71.4 per cent), as shown in Table 2. Other vessels injured included the renal, superior

Table 2. Location and Type of Arterial Injury (70 Cases)

	WITH LACERATION	WITHOUT LACERATION	TOTAL
Brachial	10	4	14
Popliteal	10	4	14
Femoral	11	1	12
Axillary	8	2	10
External iliac	3	—	3
Common iliac and inferior vena cava	3	—	3
Common iliac	2	—	2
Radial*	2	—	2
Ulnar*	1	—	1
Radial and ulnar*	1	—	1
Subclavian	1	—	1
Innominate	—	1	1
Posterior tibial, anterior tibial, and peroneal*	—	1	1
Mesenteric*	1	—	1
Abdominal aorta	—	1	1
Abdominal aorta, superior mesenteric, and inferior vena cava	1	—	1
Hepatic	1	—	1
Renal	1	—	1
TOTALS	56	14	70

* Listed since type of injury including these arteries would have caused disabling symptomatology, loss of limb or organ, or loss of life.

mesenteric, hepatic, innominate, subclavian, radial, ulnar, iliac and tibial arteries, as well as the aorta.

Of the 60 patients treated immediately after injury, reconstitution was attempted in 56. Twenty-three required closure of the laceration or excision of the lacerated area and primary anastomosis. Thirty-three patients required grafting (30 with laceration and three without laceration). The saphenous vein was utilized in 13, arterial homografts in seven, and cloth prostheses in 13 (seven nylon and six Teflon). Three patients required ligation of the vessel. Two of these had radial artery injuries and also required evacuation of the surrounding hematoma and

Table 3. Arterial Injury Without Laceration (15 Cases)

VESSEL	CASES	SPASM	THROMBUS	HEMATOMA	SOFT TISSUE EDEMA	INTIMAL TEAR
Brachial	4	3	1	2	2	—
Popliteal	4	3	1	1	2	—
Axillary	2	1	1	2	—	1
Femoral	1	1	—	1	—	—
Radial and ulnar*	1	1	1	1	—	—
Posterior tibial, anterior tibial, and peroneal	1	1	—	—	—	—
Innominate	1	—	1	1	—	1
Abdominal aorta	1	—	1	1	—	1
TOTALS	15	10	6	9	4	3

* Radial lacerated, but ulnar occluded without laceration. Listed in Table 2 as laceration only.

fasciotomy; the third had a mesenteric artery perforation from an aortogram, which necessitated excision of the ischemic bowel. One patient refused operation and later developed a Volkmann's contracture.

Fifteen arterial injuries resulting from contusion without laceration had combinations of spasm (ten cases), thrombosis (six cases), hematoma (seven cases), soft tissue edema involving a tight fascial compartment (four cases), and intimal tear without external laceration (three cases), as shown in Table 3. Only three patients were considered to have arterial spasm alone — one with a fractured ankle, and two with fractures of the proximal third of the tibia — although soft tissue edema and bony displacement could possibly have been contributory. One patient suffered an avulsed radial artery, and the ulnar artery containing a small thrombus was surrounded by a perivascular hematoma overlying a marked localized spasm. The volar fascial compartment was already open due to the injury, but the hand was cold and cadaverous.

Three vessels were occluded due to both intimal tear and thrombosis; a hematoma surrounded each vessel. None of these three patients had perceptible distal pulsations. A 40 year old man had been pinned between a trailer and an oil rig, lacerating the sigmoid colon, fracturing the ilium, and displacing an atheromatous plaque at the bifurcation of the abdominal aorta. A 19 year old youth was involved in an automobile accident in which he received a blow to the chest and head; he suffered a fracture of the mandible. Pulsations of the right carotid and subclavian arteries were absent. The chest film and a picture of the excised innominate artery are shown in Figure 1. The third case was that of a 20 year old man who was injured in an automobile accident. Fractures of the clavicle and scapula were shown by x-ray. The right upper extremity had no arterial pulsations. At operation, the right axillary artery

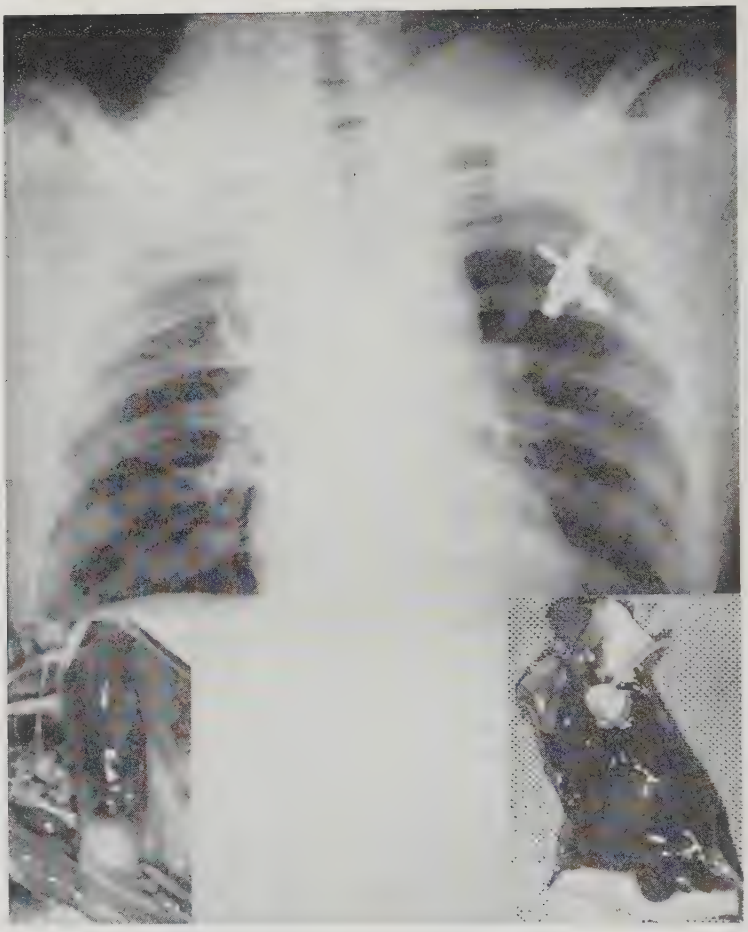

Fig. 1. Blunt trauma to chest of 19 year old male, showing widening of mediastinum. Central venous pressure catheter in left subclavian vein. *Inset at left:* Hematoma of innominate artery wall externally, with absence of pulsation, found at surgery. *Inset at right:* Sectioned innominate artery with contained thrombus and intimal tear.

was found to be occluded due to a torn intima, with a contained thrombus similar to that in the innominate artery in Figure 1.

Four of the vessels injured without laceration were in children. One four year old boy with a supracondylar fracture had a weak radial pulse which disappeared entirely after reduction of the fracture and traction. Hypoflexion, re-reduction, and release of traction all failed to improve the appearance of the cold and pulseless part in 18 hours. Operation revealed a thread-size brachial artery, surrounded by a hematoma. The second patient was the previously mentioned six year old girl with a

Fig. 2. Compartment edema in forearm and leg resulting from prolonged tourniquet application in snake bites. Both patients developed "fracture blisters." *Top:* Fasciotomy refused; patient later developed Volkmann's contracture. *Bottom:* Three-compartment fasciotomy, with return of distal pulses. Transverse "scalloping" at upper third of leg was performed as a first-aid measure before the patient's admission.

fractured ankle. The third and fourth patients (Fig. 2) were both victims of improper tourniquet application after rattlesnake bites.

Of the 67 patients who required operation either immediately or at a later time, 20 were found to have distal clots at points other than the immediate site of trauma (Fig. 3). Twenty-five patients had significant hematomas in the area of the injury or in the surrounding tissue, requiring evacuation and debridement. Two patients obtained complete relief with only evacuation of the hematomas. Twenty patients received fasciotomy because of circulatory embarrassment due to tight fascial compartments, or as prophylactic measures.

RESULTS

Thirteen failures (18.6 per cent) included three deaths (4.3 per cent), four amputations (5.7 per cent), one bowel resection (1.4 per cent), and five patients with inadequate distal pulsations resulting in disabling symptoms (7.0 per cent). The remaining 81.4 per cent have had res-

Fig. 3. Compound dislocation of right elbow, with avulsion of brachial artery, in 11 year old boy. Saphenous vein graft inserted after reduction of dislocation, debridement of wound and removal of distal clot. Complete function of joint 6 months after injury (*bottom left*), with restoration of distal arterial pulsations.

toration of arterial flow with good to excellent results; distal pulsations were present in all these patients after repair.

One death occurred after a 30 year old man had been shot through the abdominal aorta, superior mesenteric artery and inferior vena cava. All three vessels were reconstituted, but a perforation of the third portion of the duodenum was missed. In spite of two subsequent operations, the patient expired from peritonitis on the twelfth postoperative day.

The second death was that of a 40 year old man seen nine hours after blunt trauma to the abdomen. A laceration of the sigmoid was repaired. He had also suffered a fracture of an atheromatous plaque of the abdominal aorta, resulting in distal aortic thrombosis. Excision and grafting of the aorta with insertion of a Teflon prosthesis restored distal arterial pulsations, but the patient failed to respond to shock therapy and died eight hours after operation.

The third death was that of a 19 year old youth who had been involved in an automobile accident, suffering blunt trauma to the sternum, a fractured mandible, and probable head injury. The innominate artery was occluded due to an intimal tear and thrombosis (Fig. 1). Excision

and grafting with a Teflon prosthesis restored good arterial flow to both the right carotid artery and the subclavian artery, but the patient did not awaken after operation, and expired 36 hours later. The probability of intracerebral damage and the possibility of brain damage from air or thrombotic emboli after restoration of arterial continuity to the brain were not resolved because no autopsy was performed.

All four patients who required amputations had good distal pulsations after restoration of vascular continuity, but other trauma was the major factor leading to amputation. The first was a 24 year old man who suffered a shotgun injury to the femur, sciatic nerve and popliteal artery. The area was debrided and a nylon graft was inserted. Because of extreme comminution of the femur, the leg was stabilized with sandbags. One month after operation, plaster immobilization resulted in ischemia of the extremity within 24 hours. The second patient was a 23 year old woman who received a ski cable injury to the right popliteal space, which gouged out all contained muscles, nerves and veins, including a portion of the tibia and knee joint. A nylon graft was inserted after debridement. Infection developed in the extremity, and the graft was exposed. Because the outlook for a functional limb was hopeless, the extremity was amputated two weeks later.

The third patient was a 42 year old man whose arm had been caught in an industrial wringer, causing avulsion of the brachial artery, fracture of the olecranon, and disruption of many of the muscles of the arm and forearm. Multiple fasciotomies of closed spaces in both the arm and forearm, plus debridement and restoration of the brachial artery with an autogenous saphenous vein graft, resulted in good distal pulsations, but a woody, hard, painful extremity resulted. Because the arm had become useless, a midarm amputation was performed six weeks later.

The fourth amputation was in a 25 year old man with multiple fractures, and lacerations of the brachial and radial arteries. The brachial artery had been repaired previously, and the radial artery was ligated, followed by debridement and fasciotomy. However, the index finger became gangrenous, and was amputated.

Three patients required immediate reoperation. All three had distal retained clots missed at the initial operation, and all three operations were successful.

Five patients have disabling late symptoms. A femoral artery injury was caused by catheterization of this vessel for cardiopulmonary bypass during heart surgery. Repair was delayed for three days. Failure was due to a distal retained clot. The second and third patients (Fig. 2) were teen-aged boys both of whom had been bitten by rattlesnakes, one on the forearm and the other on the leg. Tourniquets were applied too tightly and allowed to remain for several hours. The patient with the forearm bite developed "fracture blisters" along with signs of ischemia. His

parents refused permission for fasciotomy, and the boy ultimately developed a Volkmann's contracture. The other boy had a three-compartment fasciotomy to the leg with immediate relief but with some residual symptoms.

The fourth patient was a 49 year old woman with mitral stenosis who underwent cardiac catheterization through the femoral artery. Closure of the arteriotomy was followed by thrombosis. Operative repair was successful initially, but in a few days the extremity became pulseless. A retained distal clot was considered to be the cause of failure.

The fifth patient sustained a femoral artery injury during varicose vein surgery. An equinus deformity developed, accompanied by a pulseless foot and causalgia-type pain. Sympathectomy for the pain, tendon transplantation, and an arterial homograft replacement of the femoral artery relieved the major complaints. However, the pedal pulses disappeared on the fifth postoperative day. A partial distal occlusion from a retained clot or thrombosis of the graft was thought to be the cause for failure.

Seven patients have some decrease in pulsations distally. Since there is no disabling symptomatology, these are classified as good but not excellent results. All had primary anastomosis with continuous suture.

Only five patients had infections at the site of arterial repair. One was the patient who died of peritonitis; the second was the girl whose leg was amputated after injury by a ski cable. The other three were in a popliteal artery repaired by an arterial homograft, an axillary artery restored by means of an autogenous vein graft, and an axillary artery reconstituted by primary anastomosis. All infections cleared, and the wounds healed without further complications.

SUMMARY AND CONCLUSIONS

The principles and many details of successful management of arterial injuries have been set forth. A review of the experience in a civilian hospital during the past ten years forms the basis for concluding that with present techniques excellent results can be obtained in more than 80 per cent of patients with a wide variety of arterial injuries and a high incidence of multiple injuries. In this group of 70 patients there were three deaths (4.3 per cent), four amputations (5.7 per cent), and inadequate restoration of circulation in five patients (7.0 per cent).

Failure of arterial repair may be due to these factors:

1. Poor surgical technique, including (*a*) stenosis of the vessel because of a narrowed anastomosis as a result of continuous suture rather than partial or complete use of interrupted sutures; (*b*) too much tension; (*c*) undue operative trauma or injudicious debridement of the vessel; (*d*)

improper application of sutures, with reliance on external thrombus rather than internal fibrin seal.

2. Incomplete total wound management, particularly inadequate debridement and failure to eliminate dead space.

3. Failure to perform distal clean-out.

4. Inadequate measures to control arterial spasm.

5. Failure to utilize the technique of fasciotomy to control complications of muscle edema.

6. Inadequate measures to prevent or eliminate shock; lack of control of total body hemodynamics before, during and after surgery.

7. Failure in management of massive and multiple injuries, particularly in elderly patients with pre-existing disease.

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Penetrating Injuries of the Inferior Vena Cava

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Despite the growing literature on the treatment of arterial injuries, comparable information on the care of great venous injuries has been slow to accumulate. Prior to 1961, there was only one report of the successful treatment of more than a single case of injury to the inferior vena cava.[5] Since that time, large series of vena caval injuries have been reported from Houston[3] and from Chicago.[6]

The purpose of the present report is to re-emphasize certain clinical features of inferior vena caval perforations, and to outline steps for the effective treatment of this potentially lethal injury. The 12 cases used for illustration of various problems have been previously tabulated[6] and Cases 1–4 and 8 have been previously described in detail.[5, 6]

CASE REPORTS

Case 1

J.M.H., Miami, 246987 (previously reported in detail[5]).

A 27 year old Negro man was shot in the abdomen with a .22 caliber

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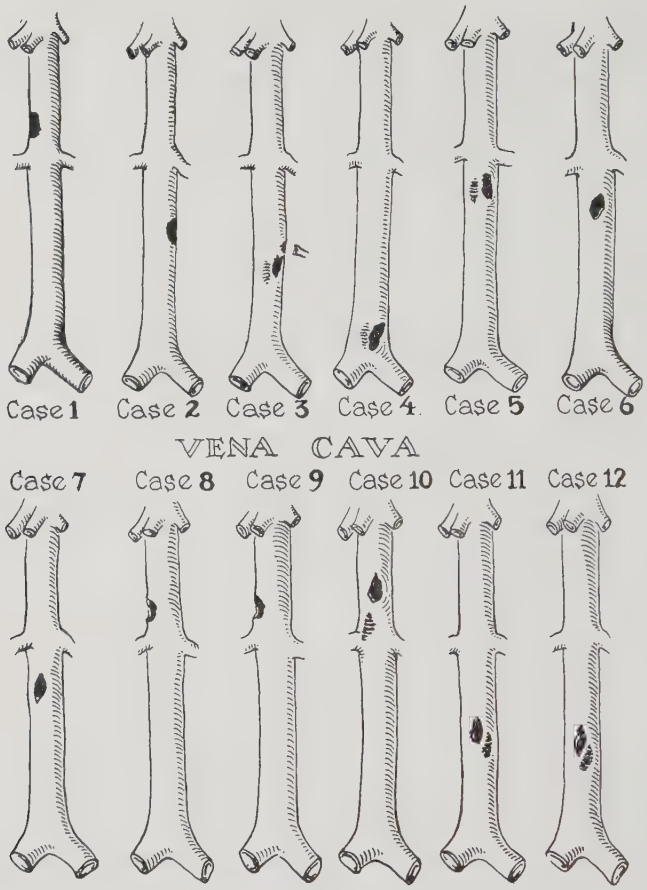

Fig. 1. The location and extent of the injuries to the inferior vena cava. The diagrams are in the same order as the case reports. (From Surgery *51:* 195, 1962.[6] By permission of the publishers.)

pistol on September 8, 1956, and was admitted to the hospital in shock. The bullet had passed through the second part of the duodenum and the head of the pancreas and had avulsed the right lateral wall of the inferior vena cava above the renal veins (Fig. 1). A large retropancreatic hematoma was present, but there was no active intraperitoneal bleeding. Massive hemorrhage, which occurred when the plane of the hematoma was entered by a Kocher maneuver (Fig. 2, *C*), was controlled with pressure and with the use of vascular clamps. The caval perforation was closed with continuous 5–0 arterial silk. The duodenal perforations were closed and the retroperitoneal space was extensively drained. Transfusions of 5000 cc. of plasma or blood were administered. Convalescence was uncomplicated, and there were no late untoward sequelae.

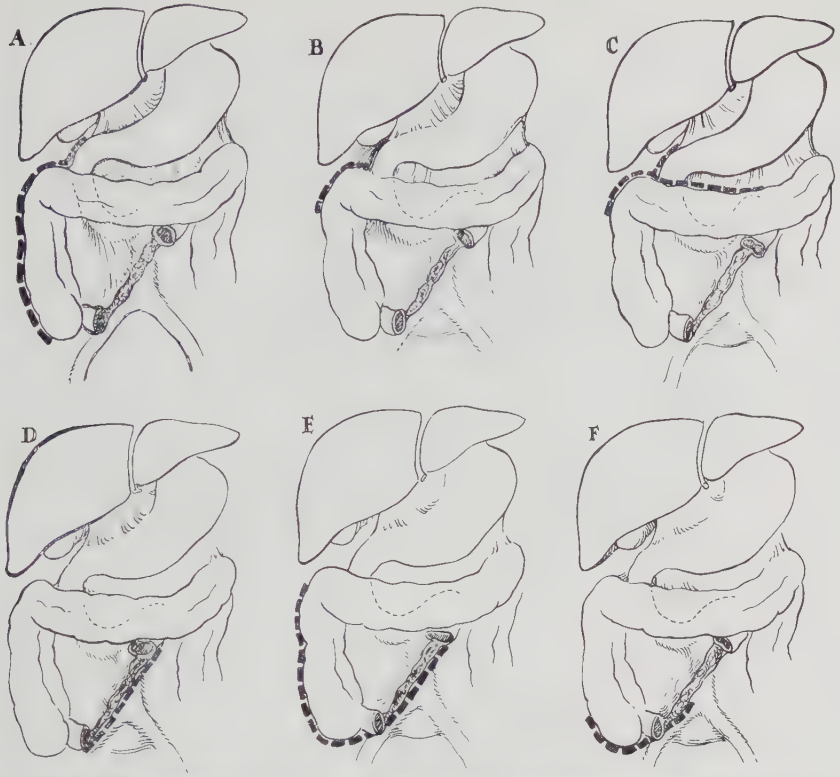

Fig. 2. Different posterior peritoneal incisions used for retroperitoneal exploration.

Case 2

J.M.H., Miami, 252952 (previously reported in detail[5]).

A 38 year old man was admitted in shock on October 19, 1956, after being shot in the abdomen with a .32 caliber pistol. At operation, active intraperitoneal bleeding had ceased, and a large retroperitoneal hematoma was found beneath the transverse colon and the mesentery of the small bowel. The bullet had passed through the stomach, the third portion of the duodenum, and between the aorta and vena cava approximately 2 inches below the renal vessels (Fig. 1), causing injury to the contiguous aortic and caval wall (Fig. 3, *A*). The vessels were approached through an incision in the base of the small bowel mesentery (Fig. 2, *D*). Abrupt hemorrhage occurred upon opening the hematoma. This was controlled with proximal and distal compression and both the aorta and vena cava were repaired with 5-0 arterial silk. The gastrointestinal perforations were closed and the retroperitoneal space drained. Transfusions of 4000 cc. of plasma or blood were given. Convalescence was rapid and follow-up revealed no late complications.

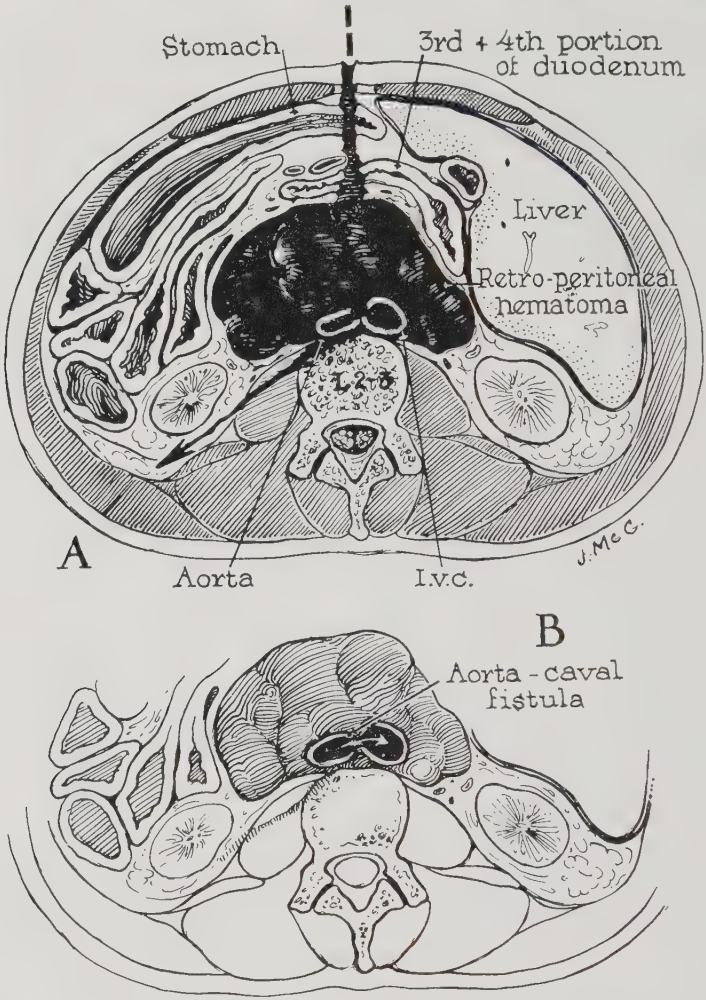

Fig. 3. *A*, The injury seen in Case 2, with laceration of the contiguous aorta and inferior vena cava.

B, Possible result if vascular injuries had not been repaired, with formation of aorta-caval fistula. Nine such cases have been reported.

Case 3

J.M.H., Miami, 266747 (previously reported in detail[5]).

A 25 year old Puerto Rican male was admitted on January 4, 1957, after being shot in the back with a .357 magnum police pistol. He was normotensive and did not appear to be critically injured. At exploration, free intraperitoneal bleeding had ceased. The bullet had passed through the jejunum and third part of the duodenum. A small hematoma elevated the ligament of Treitz. On entering the hematoma, at operation, massive

venous bleeding occurred. Broad exposure of the retroperitoneal space was obtained as shown in Figure 2, *E*. Application of Potts clamps to both iliac veins and to the vena cava above the site of injury was ineffective in controlling the hemorrhage owing to intercostal influx, and local pressure was necessary. The missile had passed through the vena cava 2 inches above the bifurcation causing 1.5 cm. lacerations of both anterior and posterior walls and avulsing a lumbar vein (Fig. 1). The caval rents were repaired with continuous 5-0 arterial silk. After suturing the intestinal perforations, the abdomen was closed without drainage. Transfusions of 5000 cc. of plasma or blood were required. Recovery was complicated by left iliofemoral thrombosis which was thought to be secondary to operative trauma to the left iliac vein. Two months after discharge, there was no evidence of venous stasis in either leg.

Case 4

U.S.V.A.R., 13588 (previously reported in detail[6]).

A 27 year old male Negro was admitted on November 2, 1956, to the U.S.V.A. Research Hospital, Chicago, three hours after having been shot in the upper midabdomen. The bullet had passed through the transverse colon, two loops of small bowel and the inferior vena cava (Fig. 1). At operation, active bleeding had ceased, but a large retroperitoneal hematoma was present.

Despite preliminary occlusion of the cava and iliac veins with tapes (Fig. 4), massive hemorrhage occurred when the retroperitoneal space was entered, owing to venous return from the intervening lumbar veins. This was eventually controlled by pressure and packing on each side of the injured vessel. An anterior caval laceration was closed with 5–0 arterial silk, and the posterior wall rolled over and closed in a similar manner. The colon and small bowel lacerations were closed in two layers, and the abdomen closed without drainage. Transfusions totaled 3500 cc. Recovery was uneventful except for a superficial wound infection.

Case 5

C.C.H., 56-65477.

A 26 year old male Negro was admitted to Cook County Hospital on October 21, 1956, six hours after being shot in the lower chest. He was unconscious from the time of injury until after admission, and his last recollection was that of leaning over an automobile hood prior to injury. He had been admitted to another hospital in shock, treated with 500 cc. plasma and Levarterenol and transferred to Cook County Hospital. Blood pressure was 90/64 and pulse 124. There was a wound of entry in the right eighth intercostal space, just lateral to the midclavicular line. The bullet, subsequently shown to be .32 caliber, could be palpated subcutaneously in the left flank, opposite the third lumbar vertebra. He

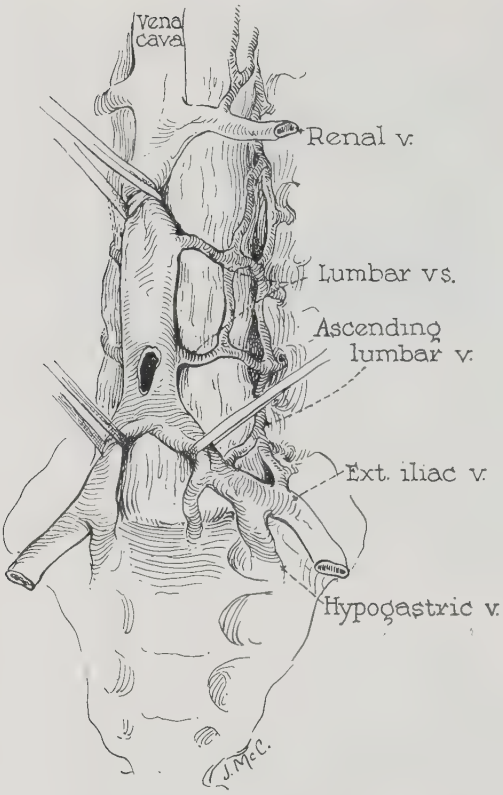

Fig. 4. Large collaterals which connect different segments of inferior vena cava. Note that tapes placed at a distance from vascular injury fail to control hemorrhage due to collateral inflow. (From Surgery, *51:* 195, 1962.[6] By permission of the publishers.)

had generalized peritonitis. No blood was present in the urine, gastric aspirate, or feces. Hematocrit was 35.

After transfusion, operation was performed eight hours after the original injury, under ether anesthesia. A long right paramedian incision was used. A large quantity of free peritoneal blood was removed, and a large retroperitoneal hematoma was noted, but active bleeding had ceased. The bullet had passed downward and to the left from the point of entry, passing through the right lobe of the liver and the anterior and posterior walls of the lower second part of the duodenum, and had entered the retroperitoneal space at the level of the second lumbar vertebra.

The retroperitoneal space was entered by mobilization of the right colon medially and with a Kocher maneuver (Fig. 2, *A*). The perforations in the duodenum were closed in two layers. The hematoma, at a somewhat lower level, was then explored further. The field was immediately flooded with blood. The hemorrhage was controlled by pressure with a stick sponge over a through-and-through perforation of the vena cava just below the renal veins (Fig. 1). Ligatures were placed above and

below the lacerations, and the vena cava doubly ligated with silk. Two Penrose drains and a Chaffin tube were used to drain the retroperitoneal and subhepatic spaces and the abdomen was closed. Transfusions totaled 3000 cc. of blood and 500 cc. of plasma.

The wounds healed primarily. Full diet was resumed by the fourth postoperative day. Drains were removed and antibiotics discontinued on the tenth day. The principal postoperative complaint was pain and swelling in both legs, particularly the left. The patient was outfitted with elastic stockings and discharged on the twenty-sixth day after operation. In June, 1960, the patient was well and without evidence of leg edema.

Case 6

C.C.H., 58–88026.

A 36 year old male Negro was admitted to Cook County Hospital on December 26, 1958, after being stabbed with a large butcher knife. He was drowsy and could be aroused only with effort. Blood pressure was 84/60, pulse 84, and respirations 20. There were two stab wounds in the right upper quadrant just below the costal margin, both lateral to the rectus muscle. He had peritonitis. No gross blood was present in the urine, gastric aspirate, or feces. After a 1500 cc. transfusion, he was anesthetized with ether and the abdomen explored through a right subcostal incision.

There was a small amount of blood in the peritoneal cavity and no active bleeding. The knife had passed sharply downward through the right lobe of the liver and the head of the pancreas. The third part of the duodenum and the mesentery of the small bowel were elevated by a large retroperitoneal hematoma. It was feared that retroperitoneal injury of the third portion of the duodenum had occurred. The retroperitoneal space was exposed with a Kocher maneuver and by mobilization of the hepatic flexure (Fig. 2, *B*). Massive hemorrhage occurred when the hematoma was entered. This was quickly controlled by stick sponge pressure immediately above and below the site of caval injury.

A 1.5 cm. laceration of the anterior wall of the inferior vena cava was demonstrated, 2 or 3 cm. below the renal veins (Fig. 1). This was repaired with 5-0 arterial silk. The posterior wall of the cava had not been damaged. Three Penrose drains and a Chaffin tube were placed about the head of the pancreas and the abdomen closed. A total of 5000 cc. of blood was administered.

The early postoperative course was complicated by right middle lobe pneumonitis, which cleared by the tenth day. Two weeks postoperatively, a pancreatic fistula developed. This closed spontaneously after wide incision and drainage of the subcostal incision on January 19, 1959. The patient was discharged in good condition on February 10, 1959. He was last seen on June 30, 1959, and was without complaints.

Case 7

C.C.H., 59–07122.

A 36 year old male Negro was admitted to Cook County Hospital on January 28, 1959, four hours after a knife fight, after having been previously seen in another hospital. He did not appear to be gravely wounded. Blood pressure was 104/60 and pulse 72. There were superficial lacerations of the supraclavicular area and of both hands. A stab wound was present half-way between the xiphoid and umbilicus through which protruded a loop of small bowel. He had generalized peritonitis. Hematocrit was 46.

Under Pentothal and ether anesthesia, abdominal exploration was carried out five and a half hours after injury, through an upper right paramedian incision. Only a few cubic centimeters of free blood was in the peritoneal cavity. The pathway of the knife had been in a posteroinferior direction. Through-and-through perforations of the jejunum, ileum and third portion of the duodenum were identified. A moderate sized hematoma elevated the third part of the duodenum.

In an effort to visualize the posterior duodenal perforation adequately, the retroperitoneal space was entered by a Kocher maneuver with extension of the peritoneal incision along the right colon (Fig. 2, *A*). When the place of the hematoma was entered, massive hemorrhage ensued. The bleeding was rapidly controlled with finger compression above and below the focus of bleeding. A 1.5 cm. laceration, 1 inch below the renal veins, was identified on the anterolateral surface of the vena cava (Fig. 1). There was no injury to the posterior vessel wall. The rent was trapped with a partially occluding Satinsky clamp and closed with continuous 5–0 arterial silk.

The multiple small bowel perforations were closed in two layers and, after bringing a Penrose drain from the retroperitoneal space out of the right flank, the abdomen was closed. Altogether, 3500 cc. of blood were given.

Convalescence was satisfactory. The patient was not febrile after the second postoperative day. The drain was removed on the seventh day, and he was discharged 11 days after operation. On February 16, 1960, he was asymptomatic and working.

Case 8

C.C.H., 60–20934 (previously reported in detail[6]).

A 23 year old male Negro was treated on March 20, 1960, for a gunshot wound of the abdomen. The bullet had passed through the right lobe of the liver, through the second part of the duodenum just below the entrance of the common duct, and through the pancreas, portal vein and inferior vena cava. The head of the pancreas was elevated by a large

retroperitoneal hematoma, but free hemorrhage from the caval and portal lacerations had ceased.

The retroperitoneal space was exposed by medial mobilization of duodenum and pancreas (Kocher maneuver) and by mobilization of the right colon (Fig. 2, *B*). When the plane of the hematoma was entered, massive hemorrhage occurred with the sudden loss of 1500 cc. of blood. This was rapidly controlled with pressure and with the tangential application of several straight Potts clamps to the general focus of bleeding. The vena caval and portal venous lacerations were identified and repaired with continuous arterial silk. The duodenal perforations were closed, and the area extensively drained. During the preoperative, operative and immediate postoperative periods, the patient received 750 cc. of plasma and 7500 cc. of blood.

He was discharged on May 3, 1960, six weeks after operation. On June 18, 1960, he was asymptomatic, without evidence of caval or portal thrombosis, and had returned to work.

Case 9

C.C.H., 60–11580.

A 20 year old white woman was admitted to Cook County Hospital on February 11, 1960, forty-two days after operative treatment elsewhere of an abdominal wound which was self-inflicted with a .38 caliber pistol. Information from the primary hospitalization indicated that the patient was not in shock on arrival. She was 4½ months pregnant. At operation, active bleeding had ceased. The bullet had passed from the midepigastrium posteriorly to the first lumbar vertebra, causing an avulsing laceration of the vena cava just above the renal veins (Fig. 1). A moderate-sized hematoma was present. There were concomitant perforations of the transverse colon, small bowel, pancreas, and branches of the superior mesenteric vein. Exposure was obtained as in Figure 2, *C*. The visceral perforations were closed and the cava repaired with arterial suture. The pancreas was drained. Postoperatively, a subphrenic abscess required posterolateral drainage, during which the right chest was accidentally entered.

Upon admission to Cook County Hospital, the patient was profoundly ill with pulse of 180 and temperatures as high as 108°. Two days after admission, she delivered a 2 pound, 8 ounce stillborn fetus. A large empyema on the right was treated with multiple aspirations and antibiotics. Pulmonary decortication of the empyema cavity was performed on March 7, 1960. A small residual empyema was treated with tube drainage on April 25, 1960. She was discharged on May 3, 1960. At the time of last examination on July 2, 1960, her condition was excellent, with no evidence of vena caval occlusion.

Case 10

C.C.H., 60–23320.

An intoxicated 27 year old Negro woman was admitted to Cook County Hospital on March 29, 1960. She had been shot three times at close range with a .38 caliber pistol four hours previously, and treated with 1000 cc. of blood and plasma at another hospital. She did not appear to be mortally wounded. Blood pressure was 140/90 and pulse 96. A superficial wound was in the left posterior axillary fold with adjacent entrance and exit punctures. Two other wounds of entry were found, one anteriorly in the fifth intercostal space just to the right of the midline, and the other posteriorly in the right flank. There was generalized peritonitis. No blood was found in the stool, gastric aspirate or urine. Abdominal x-rays revealed one bullet just below the diaphragm posteriorly, and another in the upper anterior abdominal wall.

Because of blood pressure instability, an additional 1500 cc. of blood were given through an arm cut-down. Exploration was carried out two hours after arrival through an upper right paramedian incision, with Pentothal-cyclopropane-ether anesthesia. 1500 cc. of blood were removed from the abdomen. Active bleeding had ceased. The only visceral injury was a perforating wound of the right lobe of the liver, just to the right of the midline. A small hematoma of no more than 5 or 10 cc. was seen in the posterior wall of the foramen of Winslow. No other disruption of the posterior peritoneum could be found. It was thought that the flank bullet had passed around the circumference of the body in the soft tissues, and that the only abdominal penetration had been through the inferiorly directed anterior chest wound.

During digital examination of the hematoma, massive hemorrhage occurred, which was controlled with pressure. The hepatic flexure and duodenum were mobilized (Fig. 2, *C*), and a Potts clamp placed on the vena cava above the renals, distal to the hematoma. Profuse hemorrhage continued. The incision was extended into the right chest and, after retraction of the liver to the left and superiorly, the cava was occluded above the site of injury with considerable reduction of bleeding. The bullet had passed through the vessel in a postero-inferior direction, passing through the anterior wall in the floor of the foramen of Winslow, and the posterior wall at a lower level just above the entrance of the right renal vein (Fig. 1). The anterior laceration was closed with 5–0 arterial silk. In rolling the vena cava to expose the posterior rent, the right renal vein was partially avulsed. This was repaired, in addition to the posterior bullet hole, with 5–0 arterial silk. The patient received 14,000 cc. of blood during the operation.

During closure, oozing was noted from all raw surfaces, suggesting a hemorrhagic diathesis. Postoperatively, blood drainage erupted from the

multiple abdominal drains and from the chest tube. The patient died 18 hours after operation. At autopsy, massive hemothorax and hemoperitoneum were found. The vascular wounds appeared to be well closed.

Case 11

C.C.H., 60–67350.

A 13 year old male Negro was admitted to Cook County Hospital on September 10, 1960, four hours after being shot in the abdomen with a .22 caliber rifle. Blood pressure was 100 60, pulse 80, and respirations 24. There was a wound of entry in the right upper abdomen, 2 inches above and lateral to the umbilicus. He had generalized peritonitis. Gastric contents, feces and urine did not contain blood. Hematocrit was 39. X-rays showed the bullet to the right of the fourth lumbar vertebra. A major vascular injury was not suspected.

At operation a hemoperitoneum of 750 cc. was evacuated. Active bleeding had ceased. The bullet had perforated the jejunum in six places, and had caused a through-and-through perforation at the junction of the second and third portions of the duodenum. A small retroperitoneal hematoma was found below the third portion of the duodenum. The right colon and duodenum were reflected medially (Fig. 2, *A*). Upon entering the plane of the hematoma, severe bleeding occurred which was immediately controlled by local pressure. Anterior and posterior perforations of the vena cava were encountered (Fig. 1) below the renal veins. These were closed with continuous 5–0 arterial silk. The duodenal perforations were closed in two layers, and because this resulted in constriction of the lumen, a gastroenterostomy was performed. The multiple jejunal perforations were close together and were removed by a 14 cm. resection. The retroperitoneal space was drained. A total of 1000 cc. of blood was given. The patient was discharged nine days after operation. Two months after the accident, there were no complaints.

Case 12

C.C.H., 60–70998.

A 28 year old male Negro was admitted to Cook County Hospital on September 23, 1960, after having been shot in the abdomen with a .32 caliber pistol. Blood pressure was unobtainable initially, but after 1500 cc. of blood it stabilized at 160/60. A wound of entry was found in the midline halfway between the xiphoid process and umbilicus. The patient had generalized peritonitis. Abdominal x-rays showed a chip fracture of the fourth lumbar vertebra and the bullet near the right iliac crest. A major vascular injury was suspected.

Exploration was performed through a right paramedian incision. Approximately 1500 cc. of free blood and bile were in the abdomen. Active bleeding had ceased. A large retroperitoneal hematoma elevated

the transverse mesocolon and the base of the small bowel mesentery. The bullet had projected postero-inferiorly, passing through the right lobe of the liver, the gallbladder and the head of the pancreas. The ascending colon and duodenum were reflected medially (Fig. 2, *A*). Upon entering the plane of the hematoma, massive hemorrhage occurred. This was controlled by stick sponge pressure above and below a through-and-through perforation of the vena cava (Fig. 1). The anterior wound was closed with continuous 5–0 arterial silk. With some difficulty, the cava was rolled over and the posterior laceration also was closed. Cholecystectomy, common duct exploration, and insertion of a T-tube were carried out. The retroperitoneal space was drained extensively. Total transfusion before, during and after surgery was 5500 cc.

Convalescence was complicated. The patient had a temporary partial paraplegia probably due to concussion injury of the cauda equina. Renal shutdown developed postoperatively with a rise of the blood urea nitrogen to 204, before diuresis started on the fifteenth postoperative day. After 2½ weeks, the T-tube was removed. A bile fistula developed which closed spontaneously three weeks later. The patient was discharged in good condition 6½ weeks after operation. There was no evidence of venous stasis in the lower extremities.

DISCUSSION

The high incidence of inferior vena caval injury with penetrating wounds of the abdomen has not been generally appreciated. It is probable that caval laceration occurs approximately once in every 50 cases of civilian gunshot wounds of the abdomen[2, 4, 6, 7] and once in every 300 knife wounds.[6]

Despite the difference in the weapons of assault, the location of the wounds, and the magnitude of associated injuries, the problems encountered at operation were remarkably similar in all the cases. There was, for example, no active bleeding from the injured vessel at the time of operation in any case, despite the fact that six of the 12 patients entered the hospital in shock. Although varying degrees of hemoperitoneum were present at the time of exploration, spontaneous tamponade had invariably occurred, with the formation of a retroperitoneal hematoma. Only when the hematoma was disturbed did bleeding recur, often massively to the surprise of the unsuspecting surgeon.

This feature of hematoma formation and self-tamponade, which is so characteristic of the inferior vena caval laceration, has important implications relating both to the judgment involved in exploring the retroperitoneal space and the method to be followed if retroperitoneal exploration is decided upon. Ochsner, Crawford and DeBakey[3] have cautioned against disturbing retroperitoneal hematomas if circumstances

permit. This policy may be advisable where the only injury appears to be to the vena cava. Commonly, however, retroperitoneal exploration is mandatory to treat or rule out injuries to other retroperitoneal structures which, if neglected, will lead to death. This was the situation in 11 of the 12 cases in the present series. It is interesting to speculate on the fate of the patient in Case 2, had the aortic injury not been detected and repaired. Although a fatal hemorrhage would have been the most likely outcome, it is possible that an aortocaval fistula might have developed as depicted in Figure 3, *B*. Nine such cases following penetrating trauma have been described in the world literature.

If retroperitoneal exploration is decided upon in the presence of a retroperitoneal hematoma, preliminary precautionary measures can be exercised in an unhurried and orderly manner. Exposure and lighting can be improved. Additional assistance can be called. Vascular clamps can be prepared. A large-bore needle or cut-down can be placed in the arm, since forced transfusions from the lower extremities may only aggravate the situation by flooding the field with citrated blood through the caval rent. An adequate supply of blood should be brought to the operating room. Plans can be made for the maximum exposure of the retroperitoneal space, according to the location of the injury (Fig. 2). Isolation of the great vessels above and below the site of the hematoma may reduce the ultimate extent of blood loss.

When at last the retroperitoneal space is exposed, the dissection should proceed rapidly or exsanguination may occur before the site of hemorrhage is controlled. The most effective means for immediate control of hemorrhage has been finger or stick sponge pressure upon or near the site of vascular injury, or the direct application of vascular clamps. Attempts at occlusion at any distance above and below the bleeding site do not achieve hemostasis (Case 4). The abundant lumbar collaterals[1] interconnect the different levels of the vena cava so effectively (Fig. 4) that hemorrhage may be almost as severe as with the cava open. The difficulties with hemorrhage can be appreciated by the fact that an average of 5200 cc. of blood or plasma was given to these 12 cases.

Suture repair of the caval rent was performed in all except Case 5, in which ligation was done. Subsequent complications attributable to the venous repair occurred only in Case 3, in which ileofemoral thrombosis occurred. In the two knife wounds (Cases 6 and 7), the blade lacerated only the anterior wall. With gunshot wounds there was either a double perforation (Cases 3, 5, 10, 11, 12) or a lateral avulsion (Fig. 1) with loss of tissue (Cases 1, 2, 4, 8, 9). In the cases with injury above the renal veins (Fig. 1), suture repair is mandatory since caval ligation at this level leads to death.[8]

In the cases presented, vena caval laceration never occurred as an isolated injury, a minimum of one, a maximum of four and an average

of 2.4 other organs being involved. These included associated injuries to the liver, pancreas, stomach, duodenum, jejunum, ileum, colon, gallbladder, aorta, portal vein and superior mesenteric vein. These injuries necessitated extensive use of intra- or extraperitoneal drains in all but two cases, and contributed to serious complications which included pancreatic fistula, subphrenic abscess, empyema, biliary fistula, and acute renal failure. The high survival rate of 11 of 12 patients was undoubtedly partly due to the fact that all the patients were less than 38 years old and in previous good health.

SUMMARY

Clinical summaries are presented of 12 patients who sustained penetrating wounds of the inferior vena cava in addition to other visceral injuries. The wounds were inflicted by gunshot in ten cases and by knife lacerations in two. Eleven of the 12 patients survived.

Injury to the inferior vena cava as a result of penetrating wounds of the abdomen is more common than is generally appreciated. Characteristically, hemorrhage from the venous injury spontaneously ceases as the result of retroperitoneal tamponade. Exsanguinating hemorrhage is frequently provoked by exploring the retroperitoneal hematoma at the time of operation.

If a retroperitoneal hematoma is found at operation in the locality of the great vessels, precautions should be taken before the retroperitoneal space is explored. These include provisions for rapid transfusion, adequate or extended exposure, and instruments for vascular reconstruction.

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Traumatic Pancreatitis, an Increasing Problem

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The recognition and management of traumatic pancreatitis is a perplexing and uncommonly reported problem. Prior to 1900, early diagnosis was rarely made and treatment usually awaited the development of an abdominal mass or pseudocyst.[5] Even in 1961, Fogelman[3] was able to find only 300 cases in the world literature, principally in the form of individual case reports. During the Korean and Second World Wars, Poole[6] found that the number of reported cases was small, with a mortality rate of 22 to 56 per cent.

In civilian practice, pancreatic injury occurs with sufficient frequency to be considered an important entity. For example, 20 cases of traumatic pancreatitis have been treated at Colorado General Hospital in the past ten years. In 14 patients the injury was caused by blunt trauma and in the other six by penetrating wounds. These cases have been critically examined with particular attention to the clinical features, and to the type and effectiveness of surgical therapy.

TYPE OF INJURY

Fourteen patients sustained blunt trauma, and six cases resulted from penetrating injury. With three exceptions, the blunt trauma was sustained in automobile accidents. Mathewson[5] reported 17 cases, of which nine were due to blunt trauma and the remainder to penetrating injury. In Fogelman's series,[3] 30 of the 33 cases reviewed had penetrating type of injury, and in three the injury was of the blunt type. In the present series all six penetrating injuries of the pancreas were caused by gunshots.

ASSOCIATED INJURIES AND SURVIVAL

Associated injuries were frequent, and the treatment of these often took precedence over treatment of the pancreatitis. In the series of cases under review here, the liver, spleen and bowel were the most commonly injured organs (Table 1). The significance of associated injuries becomes

Table 1. Associated Injuries

Liver	9
Spleen	7
Bowel	8
Chest	3
Stomach	1
Great vessels	1
Diaphragm	2
Head and skeleton, etc.	10
Total	41

apparent when one evaluates the mortality figures. When three or more associated organs were involved (eight cases), the mortality rate was 100 per cent. If the injuries involved fewer than three organs, the survival rate was 92 per cent (12 cases). Ten of 14 patients with blunt trauma survived, while only one of six survived penetrating injuries.

Rupture of the duodenum was an associated injury in six patients, five of whom succumbed. Penetrating injuries (gunshots) were responsible in three cases. Early exploration and repair of the duodenal injury was carried out in these cases. All three patients died of shock and multiple injuries on the day of admission or shortly thereafter. The two fatalities after duodenal rupture and pancreatitis from blunt trauma represent diagnostic errors and delayed and inadequate treatment. In both, the vague and uncertain findings on admission were misinterpreted until more obvious signs of peritonitis led to laparotomy 24 and 48 hours later. Shock inadequately treated was the immediate cause of death. In one of these cases, the serum amylase concentration was high on admission. It is regrettable that more significance was not attached to this observation.

COMPLICATIONS

The most common complication in this group of cases was infection which involved the local area, with extension to the subphrenic spaces in three instances and to the general peritoneal cavity in two. One patient had a grossly infected wound, and in three others major pulmonary complications resulted from infection, hemorrhage and atelectasis. In no instance was the infection directly and singly attributable to pancreatic

injury. Rather, multiple injury, extensive hematoma and tissue damage were equally important, particularly when the bowel was penetrated. Regardless of this, the high incidence of infection is noteworthy and demanding of consideration.

Pseudocyst formation was the most common single complication of traumatic pancreatitis (five cases) (Table 2 and Cases I and II). In

Table 2. Complications

Fistula	2
Subphrenic abscess	3
Peritonitis	2
Wound	1
Gastrointestinal hemorrhage	4
Pseudocyst	5
Pulmonary	3
Other	3
TOTAL	23

three cases, pseudocysts formed when no drainage was instituted. There were two instances of pseudocyst formation after early drainage. Internal drainage into defunctionalized loops of small bowel was the method used to cure each of these cases.

Chronic fistulas from the pancreas developed in two cases in which drains were placed to badly damaged glands. In one the fistula closed spontaneously, and in the other the sinus tract extended from the main pancreatic duct to the exterior. This was the only demonstrable drainage route from the distal part of the pancreas. After drainage for seven months, the sinus tract was anastomosed to the jejunum.

In Table 2 it can be seen that, after infection and pseudocyst formation, massive gastrointestinal hemorrhage was the most common complication in this series of cases. Subtotal gastric resection was performed in two cases and a vagotomy and pyloroplasty were done in the third case, with control of hemorrhage and survival. In the fourth patient, nonoperative treatment ended fatally. Duodenal and gastric ulceration was not established in any of these cases (laparotomy in three and autopsy in one). Mucosal bleeding in the stomach was active in the three patients examined at laparotomy, and this appeared to be the site of bleeding in the fourth patient at autopsy. Although the exact relationship between pancreatic and gastric function has not been clarified, there are experimental situations which indicate that such relationships do exist.[2, 4, 7] The intriguing possibility that pancreatic trauma and subsequent inflammation may underlie the development of hemorrhagic lesions of the stomach other than ulcer finds support in these cases. Of considerable interest is the fact that, in the patient subjected to pyloro-

plasty and vagotomy, the bleeding was observed to stop within five minutes after section of the vagus nerves. It is apparent that massive gastrointestinal hemorrhage must be treated aggressively, and that reasonable success can be expected by standard operative approaches despite the unfavorable circumstances under which operation is carried out.

DIAGNOSIS

In the authors' opinion, the most important factor in the diagnosis of traumatic pancreatitis is an awareness on the part of the examining surgeon that it may exist in any patient who has had trauma to the abdomen.

Diagnostic aids such as serum amylase determination, peritoneal aspiration and radiography have been used with variable success. Measurement of serum amylase concentration was carried out in 12 of the 20 cases, and was considered helpful in arriving at a tentative preoperative diagnosis or in following the progress of the disease in ten instances (Table 3). Inasmuch as this test was not routinely applied, considerable bias probably entered into the selection of the patients to be examined by this method, and the results are undoubtedly skewed toward a high correlation between the actual presence of pancreatitis and elevated serum amylase concentrations. Despite this, some interesting facts emerge from examination of the available data summarized in Table 3. In six patients, serum amylase was measured at the time of admission, before operation and within 24 hours of injury. In four instances, abnormal levels were found. The other two patients subsequently were found to have high enzyme concentrations. In fact, all 12 patients with traumatic pancreatitis on whom amylase determinations were made had abnormally high enzyme concentrations at some time in the course of the disease. Of particular interest is the close correlation between the rise and fall of serum amylase and the waxing and waning of the disease.

In seven instances, secondary rises in serum amylase concentrations were associated with persistent or reactivated inflammatory processes requiring further drainage in each case. Amylase determinations on aspirated retroperitoneal fluid collections were very high in two cases, and the amylase content of free peritoneal fluid in one case was moderately elevated.

The four-quadrant peritoneal tap, as described in more detail elsewhere in this volume,[1] was performed in six cases. Bloody or turbid fluid was aspirated from five patients. The diagnosis of perforated viscus was suggested, but in no instance was the diagnosis of pancreatitis made on the basis of this procedure.

Table 3. Serum Amylase Determinations* in 12 Cases of Traumatic Pancreatitis

TYPE OF INJURY	TIME OF FIRST DETERMINATION	TYPE OF SURGERY	VALUE OF FIRST DETERMINATION	LENGTH OF TIME ELEVATED	RANGE OF VALUE	SECONDARY ELEVATION OR PROLONGED ELEVATION	IMPLICATION OF SECOND RISE	RESULT	ABD. OR RETRO-PERIT. FLUID
Blunt	1st day	External drainage and later drain. of retroperitoneal mass	904	14 days	376–904	Yes	Reaccum. of retroperitoneal mass	Alive	
Blunt	Before surg. on day of injury	External drainage	74	15 days	146–330	None		Alive	
Blunt—adm. 4 wks. after injury	40 days after injury	Internal drainage	1197	16 days	174–1243	None		Alive	
Blunt	3 days after injury	Splenectomy and 3 days later external drainage	457	42 days	155–457	Yes—5 days later	Mass RUQ; necrotic pancreas	Alive	
Gunshot	Before surg. on day of injury	External drainage	355	7 days	160–355	None		Alive	
Blunt	1st day in hospital	External drainage and later secondary drainage	43	5 days	43–575	Yes—10 days later 575	Large retroperitoneal accum. around pancreas	Died	
Blow to abdomen 2 weeks prior to adm.	1st day in hospital —2 weeks after injury	External drainage	244	7 days	202–244	None		Alive	Retro-perit. fluid 927 U.
Blunt	Before surgery on day of injury	External drainage and later secondary drainage	813	26 days	370 832	Yes—8 days later 635	Large subphrenic and retroperitoneal accum.	Alive	Abd. fluid 325 U.; retroperit. fluid 1700 U.
Gunshot	2nd day	Drainage and ligation of pancreatic duct	976	7 days	168–976	None		Died	
Gunshot	2nd day after injury	Drainage and ligation of pancreatic vessel	620	3 days	264–620	None		Died	
Blunt	1st day	Repair of rupt. duod.; drained retroperitoneal mass	268	4 days	268–770	None		Died	
Blunt	Day of admission	Repair of duodenum and drainage	87	18 days	87–565	Yes	Acute pancreatitis	Alive	No

* Normal Values, 65 to 135 Somogyi Units.

Radiography was of little help in the early diagnosis of traumatic pancreatitis. In some cases, absence of psoas shadows in plain roentgenograms of the abdomen sometimes suggested retroperitoneal pathology. If a pseudocyst formed or enlargement of the gland resulted from inflammation, displacement of surrounding viscera demonstrable by barium contrast studies was helpful confirmatory evidence (Case IV).

In many cases, the most direct and appropriate means of establishing the diagnosis of traumatic pancreatitis was careful examination of the gland at laparotomy. In the present series of cases, there were 11 survivors and eight deaths. In eight of the survivors, the pancreatic injury was recognized at an early laparotomy and extensive drainage of the retroperitoneal space was provided. The remaining three patients were admitted to the hospital with established complications. If the possibility of pancreatic injury exists, the lesser omental sac should be widely opened and the entire gland visually inspected. Edema, fat necrosis, ecchymosis, minor disruptions of the parenchyma, as well as more gross pathologic changes, should be noted in order to allow early rather than late treatment of a potentially fatal condition. Measures to be instituted at operation are discussed below.

TREATMENT

Operative

The importance of laparotomy and careful examination to determine the presence of and extent of pancreatic injury has been discussed above. Particular emphasis has been placed upon opening of the lesser omental sac and visualization of the pancreas as a means of establishing the diagnosis. This recommendation stems from the fact that early diagnosis with provision for drainage of pancreatic juice was the only form of management that allowed recovery of a high percentage of patients. Eight of the 11 survivors were handled in this manner. The three other survivors entered the hospital with late complications of pancreatic injury which presumably could have been prevented had the process been recognized and treated at the time of injury. All three had laparotomy, but without recognition of the full extent of the injuries. Further emphasis of the critical importance of early diagnosis and correct assessment of the nature of the lesions comes from examination of the fatalities in this series. The two patients with blunt trauma to the abdomen which resulted in rupture of the duodenum and pancreatitis received inadequate and late treatment. Both could have been saved. One of the patients who died after a gunshot wound probably would have survived had a large retroperitoneal hematoma been explored and duodenal and pancreatic penetrations been recognized at the initial laparotomy. Lesions of the

aorta and vena cava were successfully controlled. The conclusion seems inescapable that unrecognized severe traumatic pancreatitis is a highly fatal lesion, particularly when it is accompanied by disruption of pancreatic tissue and leakage of pancreatic juice. Both local and systemic sequelae can be fatal if diagnosis and treatment are delayed.

The operative management of a damaged gland depends to a large extent upon the type of the lesion. The most common type of pancreatic injury in this series was contusion and fragmentation, reflecting the preponderance of blunt trauma sustained in automobile accidents. Identification of anatomical landmarks was difficult, and often there was active bleeding along the superior border of the gland. Where possible, devitalized fragments were excised. In most of the cases, bleeding was controlled and multiple drains were placed to the damaged glands. On review of these cases, this appeared effective, as shown by the fact that in six of eight patients in whom drainage of the pancreas was provided, the injuries healed without further complications. Conversely, three patients had laparotomy without recognition of pancreatic injury, and all developed pseudocysts or abscesses of the pancreas and adjacent areas.

In none of the patients in this series was any type of duct reconstruction feasible. In fact, identification of the major duct system was not mentioned in the descriptions of operations. In the contused glands, no dissection of ducts seemed warranted, and in the more severely damaged glands tissue destruction and maceration precluded any accurate identification.

As indicated above, penetrating injuries of the pancreas were highly fatal in this experience. This is a reflection of the severity and multiplicity of injuries and in some cases failure to adequately evaluate and treat pancreatic lesions. The authors find no evidence that debridement and provision of wide drainage was any less effective in this group of patients than in the other forms of disease. It would appear even more imperative, since free drainage of pancreatic juice into the peritoneal and retroperitoneal tissues is inevitable.

Postoperative

The details of postoperative management of these critically ill patients will depend upon the particular variety of pathology and the patient's response to injury. Disregarding other injuries, traumatic pancreatitis in itself demands heavy appropriate antibiotic therapy, anticholinergic medication to suppress pancreatic and gastric secretion, nasogastric suction, and blood volume, water and electrolyte replacement. Plasma losses and sometimes pancreatic juice leakage may be great for a few days after injury. Persistent shock usually reflects inadequate blood volume replacement.

ILLUSTRATIVE CASES

Case I. A 22 year old white man was admitted to the emergency room of Colorado General Hospital shortly after an automobile accident in which the steering wheel had struck the upper abdomen. His chief complaint was abdominal pain. Physical examination disclosed a rigid abdomen. X-rays were negative, as was the four-quadrant peritoneal tap. His vital signs and hematocrit were within normal limits. Eighteen hours later, the vital signs were stable, and repeated four-quadrant peritoneal taps were negative. However, his abdomen remained rigid. Because of this persistent sign of peritoneal irritation, exploration was undertaken. Serum amylase before exploration was 904 Somogyi units. Multiple shallow lacerations of the liver were found, with a very large retroperitoneal hematoma involving the pancreas. "Soap" deposits were present. Multiple drains were placed in the retroperitoneal area of the pancreas.

Postoperatively, the patient did well for several days, with a progressive fall in the serum amylase to 376. However, he then became feverish and had leukocytosis and abdominal pain. Subsequently, a large infected pancreatic cyst was drained externally. He again improved until the following day, when a massive upper gastrointestinal hemorrhage occurred which failed to respond to conservative measures. Exploration was again undertaken. Diffuse gastric bleeding was found, and a vagotomy and pyloroplasty were performed. No further bleeding occurred. The patient developed a pancreatic fistula that persisted for 17 weeks, when its drainage was diverted into a Roux-Y loop of bowel. He has been well since.

Comment. This case illustrates several points, the most important one being the institution of adequate drainage. Although the pancreatic injury and pancreatitis were recognized and drains were placed, these were insufficient. The peritoneum was not adequately incised to allow drainage from the retroperitoneal space. The incidence of gastrointestinal bleeding following this type of trauma is significant, and must be treated vigorously if the patient is to survive.

Case II. A 17 year old white youth was admitted to Colorado General Hospital complaining of abdominal pain following an automobile accident. He had moderate tenderness in the abdomen, maximum in the left flank. An intravenous pyelogram revealed displacement of the left kidney laterally and obliteration of the left psoas shadow. The serum amylase was 74 Somogyi units, and the hematocrit was 39. The vital signs were stable, and a tentative diagnosis of retroperitoneal hematoma was made. However, 8 hours later the hematocrit was 32, and the abdominal tenderness had increased. A small amount of blood was found on peritoneal tap. Abdominal exploration revealed a large retroperitoneal hematoma involving the left gutter, pancreas and splenic pedicle. Multiple drains were inserted. Five units of blood were given. The serum amylase rose to 330 Somogyi units and remained at this level for 7 days. It then gradually diminished. The patient was maintained on nasogastric suction and anticholinergic drugs for 7 days. His recovery was uneventful.

Comment. This case illustrates the necessity for recognition of pancreatic contusion and adequate drainage. Postoperatively, decom-

pression of the gastrointestinal tract with suction and suppression of secretion with anticholinergics are important. Both should be continued until the pancreatitis subsides.

Case III. A 6 year old boy had fallen some 6 weeks before admission. He did well initially, but suddenly collapsed a few days later. Shock was corrected, and an exploratory celiotomy was performed in another hospital. Liver lacerations were sutured. He then convalesced satisfactorily until 10 days before admission to Colorado General Hospital, when gradual enlargement of the abdomen was noted. Radiographic studies including gastrointestinal series and barium enema revealed displacement of the splenic flexure of the colon. Liver function tests were normal. The amylase was 197 Somogyi units. Exploration was performed, and a large pseudocyst of the pancreas was found. The cyst was drained into a defunctionalized segment of jejunum. The postoperative course was uneventful.

Comment. This case demonstrates the importance of recognition of pancreatic trauma in association with other intra-abdominal trauma, and the necessity for early drainage. It also illustrates that, if drainage is not performed early following pancreatic injury, pseudocyst formation will sometimes occur.

Case IV. A 37 year old white man was transferred to Colorado General Hospital 3 days after an automobile accident in which he suffered blunt abdominal trauma. A ruptured spleen was removed shortly after the accident, in another hospital. He began to complain of abdominal pain about 3 weeks after the injury. The serum amylase was 454 Somogyi units on admission. Roentgenographic examination of the upper gastrointestinal tract revealed deformity of the duodenum due to an extrinsic mass medially. Multiple infusions of blood, plasma and norepinephrine were necessary to maintain blood pressure during this period.

Laparotomy was performed, and a large retroperitoneal hematoma was found, associated with pancreatic necrosis. Multiple drains were placed. The patient recovered, and was doing well one year later.

Comment. This case clearly emphasizes the importance of early recognition of pancreatic trauma and effecting prompt adequate drainage. The near-fatal hypotensive episode was due to unrecognized pancreatic necrosis with massive local blood and plasma loss.

DISCUSSION

In any consideration of trauma to the pancreas, it is impossible to make a clear distinction between the direct effects of the trauma itself, inflammatory changes in the gland initiated by trauma, obstructive sequelae resulting from trauma, and the effects of leakage of pancreatic juice into the parenchyma of the gland and the surrounding tissues. Such

a distinction, if possible, would in all probability serve little purpose, since to a greater or less degree all or at least more than one of the above factors contribute to the overall problem. The 20 cases available for this study constitute a rather small but informative body of material representing many variations on two themes.

First, the diagnosis is difficult to make preoperatively, and even at laparotomy major pathologic processes may escape detection. If the diagnosis is not made early, the severe forms of the disease will terminate fatally, and even the lesser pathologic processes will progress and lead to complications of major importance. Second, wide drainage of the damaged pancreas and retroperitoneal area is the single most effective measure that can be instituted aside from establishment of the diagnosis. Provision of external drainage minimizes the local irritative effects of pancreatic juice and secondarily gives some relief from the complications attending duct system damage and obstruction. Retroperitoneal sepsis and pseudocyst formation can be controlled or prevented in a high percentage of cases if early adequate drainage is instituted.

It will be apparent to the reader that the problem of early diagnosis and to some extent problems of management of these cases indirectly stems from the fact that blunt abdominal injury accounted for approximately two-thirds of the cases. Previous reported experience with penetrating pancreatic injury and the extensive study of nontraumatic pancreatitis have not provided satisfactory solutions to the problems met. In fact, conservative approaches to traumatic pancreatitis along lines followed in dealing with ordinary forms of pancreatitis have solved few problems and have led to disappointment and disaster. The number of automobile accidents is increasing, and it seems certain that pancreatic injury will become more frequent and of greater importance in the practice of surgery.

Finally, an additional lesson and generalization are clearly apparent from this study. In no other disease will persistence and pursuit be better rewarded. Seemingly unending complications, crises, and long intervals of stalemate can eventually be resolved, with complete recovery. The pancreas wants to heal.

SUMMARY

Twenty cases of traumatic pancreatitis are presented, with discussion of diagnostic techniques, operative and nonoperative therapy, prognosis and complications, along with four case histories to illustrate and emphasize the various types of injuries, complications and treatment.

Establishment of the diagnosis is difficult, and failure to make an early diagnosis was responsible for some fatalities and complications. Wide drainage of the pancreas and retroperitoneal area is recommended.

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Retroperitoneal Rupture of the Duodenum Due to Nonpenetrating Trauma

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Retroperitoneal rupture of the duodenum due to blunt trauma has been reported with increasing frequency. At the present time, the majority of these injuries result from automobile accidents, most frequently by forceful impact against the steering wheel. Unless the design of the steering mechanism is radically altered, even greater numbers of these serious injuries can be anticipated as a result of more cars and higher speed limits.

Because of the relative rarity of this lesion, no single surgeon or clinic has had sufficient experience with the condition to allow sound appraisal of methods of diagnosis and management, to say nothing of prevention. Various authors have reported limited experiences, and periodically the material has been collected and reviewed, most recently by Cohn et al. in 1952.[1] The present review and analysis was undertaken to report nine additional cases (Appendix), and from this as well as other experiences reported since 1952 to formulate reasonable approaches to diagnosis and treatment.

MATERIAL AND METHODS OF ANALYSIS

This review and discussion has been confined to the entity of ruptured duodenum due to blunt trauma, in which the retroperitoneal portion of the duodenum was involved. In some instances the tears of the duodenum communicated secondarily with the peritoneal cavity. Such cases have been included. Specifically excluded, not only in our own case selection but in those cases reviewed from the literature, have been those injuries in which only the peritoneal surface of the duodenum was involved, as well as penetrating wounds caused by stabbing or gunfire.

The tabulation of cases in Table 1 includes the age, sex, mechanism of

Table 1. Published Reports 1951–1961 Inclusive, Plus Nine Additional Cases

AUTHOR	AGE/SEX	INJURY	HOSPITAL	SURGERY	LESION/LOCATION	OPERATION	RESULT	ASSOCIATED INJURIES AND COMMENTS
Harrold, 1951	63/M	Automobile steering wheel	1½ hours	10 hours	Laceration, 3 cm., posterior, first part	Closure and drainage	Survived	
Estes, 1952	32/M	Abdominal blow	½ hour	4 hours	Laceration, complete, 3rd part	End-to-end anastomosis; drainage	Survived	
Pirkle, 1953	26/M	Automobile accident	6 hours	7 hours	Tear, second part	Closure both ends. Cholecystoduodenostomy and gastroenterostomy	Survived	
Keith, 1954	23/M	Automobile steering wheel	7 hours	18 hours	Laceration, third part	Closure and drainage	Survived	
Keith, 1954	31/M	Automobile steering wheel	5 hours	7 months	Perforation, third part. Later obstruction and retroperitoneal accumulation	Drainage large intra-abdominal accumulation, LLQ	Survived	
Stransky, 1954	29/M	Automobile steering wheel	2 hours	10 hours	Tear, 3 cm., third part	Closure and drainage	Survived	
Kelly	31/M	Automobile steering wheel	6 hours	18 hours	Laceration, three-fourths of circumference of fourth part	Closure and drainage	Survived	
Kelly, 1954	14/M	Fell against fence post	4 hours	15 hours	Laceration, seven-eighths of circumference of third part	Closure and drainage	Survived	
Strode and Gilbert, 1955	13/M	Fell against curb	20 hours	Immediate	Perforation, posterior wall, third part	Closure and drainage	Survived	
Strode and Gilbert, 1955	39/M	Automobile accident	3 hours	Immediate	Tear, third part	Resection and duodenojejunostomy; implant of common bile duct; oversewed pancreas	Survived	Traumatic pancreatitis

Table 1. Published Reports 1951–1961 Inclusive, Plus Nine Additional Cases—*Continued*

AUTHOR	AGE/SEX	INJURY	HOSPITAL	SURGERY	LESION/LOCATION	OPERATION	RESULT	ASSOCIATED INJURIES AND COMMENTS
Rothchild, 1956	49/M	Horse kick	10 hours	12 hours	Laceration, three-quarters of circumference, second part	Closure and drainage	Survived	
Hansen, 1957	17/F	Horse kick	1 hour	21 hours	Complete transection, third part	Closure open ends of duodenum; gastrojejunostomy and drainage	Survived	Duodenal fistula—closed 16th postoperative day; traumatic pancreatitis
Patton, 1957	37/M	Falling object hit epigastrium	22 hours	26 hours	Laceration, 2 cm., second part	Closure; no drainage	Died	No autopsy
Patton, 1957	37/M	Fell from first-story scaffold	1 hour	12 hours	Almost complete transection at junction second and third parts	Closure and drainage	Survived	
Hanley, 1958	19/M	Crushed against wall	Several hours	Several hours later	Laceration, two-thirds of circumference, junction of second and third parts	Closure and drainage	Survived	
Webb, 1958	—	Automobile steering wheel	—	—	Complete laceration at ligament of Treitz	—	Died	
Webb, 1958	—	Automobile steering wheel	—	—	—	—	Died	
Webb, 1958	—	Automobile steering wheel	—	—	—	—	Survived	
Mabry, 1959	30/M	Automobile steering wheel	—	—	Perforation, second part	Closure and drainage	Survived	Duodenal fistula
Boggs, 1959	43/M	Automobile	6 hours	17 hours	Complete transection, third part	Closure distal end of duodenum; duodenojejunostomy, proximal end	Survived	Second admission later for abdominal pain and vomiting; adhesions over anastomosis

Table 1. Published Reports 1951–1961 Inclusive, Plus Nine Additional Cases—*Continued*

AUTHOR	AGE/SEX	INJURY	HOSPITAL	SURGERY	LESION/LOCATION	OPERATION	RESULT	ASSOCIATED INJURIES AND COMMENTS
de Niord, 1959	45/M	Automobile steering wheel	Immediate	Immediate	Laceration, 3 cm., junction second and third parts	Closure and drainage	Survived	
de Niord, 1959	50/F	Automobile steering wheel	Several hours	(1) 24 hrs (2) 48 days	Laceration, fourth part of duodenum at ligament of Treitz	(1) Drainage retroperitoneal area near ligament of Treitz (2) Resection and duodenojejunostomy	Died	Patient too ill for definitive procedure at first operation
Adnopoz, 1961	51/M	Slipped in bathtub	2 weeks	Immediate	Laceration, three-quarters of circumference, second part	Closure and drainage	Survived	Also had acute appendicitis; swallowed a fish-bone 2 days before admission
Johnson, 1957	37/M	Blunt blow; exploding emery wheel	Immediate	Immediate	Three lacerations of retroperitoneal duodenum	Closure of lacerations of duodenum and liver, cholecystectomy drainage of retroperitoneal area	Survived	Lacerated liver; gallbladder severed from cystic duct
Johnson, 1957	39/M	Fell against hydrant	Immediate	4½ hours	4-cm. laceration, second part of duodenum	Closure and drainage	Survived	Lacerated liver; duodenal fistula third postoperative day, closed in 9 weeks
Johnson, 1957	Adult/M	Automobile steering wheel	Immediate	Immediate; second operation 8 days later	Perforation, second part	(1) Exploratory laparotomy; retroperitoneal area not explored (2) Resection of perforation and end-to-end anastomosis, drainage	Died	Perforated duodenum not seen at first operation; patient died 24 hours after second operation
Johnson, 1957	5/M	Blow—heavy box fell on patient	2 hours	8 hours	Almost complete tear, fourth part	Anastomoses, proximal duodenum to jejunum and distal end of duodenum to stomach; drainage	Survived	
Johnson, 1957	16/M	Kicked by horse	—	24 hours	Site not known	Exploratory laparotomy; drainage of retroperitoneal area; jejunostomy	Survived	Duodenal fistula

Table 1. Published Reports 1951–1961 Inclusive, Plus Nine Additional Cases—*Continued*

AUTHOR	AGE/SEX	INJURY	HOSPITAL	SURGERY	LESION/LOCATION	OPERATION	RESULT	ASSOCIATED INJURIES AND COMMENTS
Cleveland and Waddell, 1962	21/M	Automobile next to driver	3 hours	11 hours	Laceration, third part; contused pancreas; large retroperitoneal hematoma over pancreas	Closure and drainage; resection of contused ileum	Survived	Traumatic pancreatitis
Cleveland and Waddell, 1962	45/F	Automobile steering wheel	21 hours	2½ days	Laceration, 1 cm. diameter, second part	No. 20 Pezzar catheter inserted into duodenum; retroperitoneal space drained	Survived	Duodenal fistula
Cleveland and Waddell, 1962	5/M	Blow to abdomen	48 hours	6 days	Laceration, 10 cm., fourth part, at ligament of Treitz	Duodenojejunostomy; drainage	Survived	
Cleveland and Waddell, 1962	45 F	Automobile steering wheel	4 hours	36 hours	Laceration, three-fourths of circumference, junction second and third parts; contused pancreas, large retroperioneal hematoma	Resection 2 cm. of duodenum; end-to-end duodenoduodenostomy; drainage	Died	Traumatic pancreatitis at autopsy
Cleveland and Waddell, 1962	24 F	Automobile steering wheel	Immediate	17 hours	Laceration, third part	Closure and drainage	Survived	
Cleveland and Waddell, 1962	1/F	Blow to abdomen	5 days	7 days	Complete transection, third part	Duodenojejunostomy; drainage	Survived	
Cleveland and Waddell, 1962	12 M	Automobile ran over	Immediate	7 hours	Laceration, second part	Closure and drainage; splenectomy and closure of laceration of liver	Survived	Duodenal fistula and subphrenic abscess; also had ruptured spleen and liver
Cleveland and Waddell, 1962	30, M	Automobile steering wheel	Immediate	18 days	Perforation, third part	Drainage; duodenojejunostomy	Survived	Fistula persisted for 10 days
Cleveland and Waddell, 1962	42 M	Automobile steering wheel	3 hours	7 hours	Laceration, third part	Closure and drainage	Survived	Traumatic pancreatitis

injury, site of injury to the duodenum, interval between injury and hospitalization, interval between injury and surgical treatment, type of operative procedure carried out, and results. Other data such as the type and incidence of associated injury, complications after duodenal rupture, and in a few instances ascribable causes of death are available from this analysis and have been presented in the discussion.

RESULTS AND DISCUSSION

Mechanism of the Injury (Table 2)

The mechanism of injury to the duodenum is exceedingly variable. Any type of blow to the abdomen can result in rupture of the duodenum. Sometimes relatively light blows produce such injury. It is of interest that in Cohn's collected cases[4] there were only four instances in which the injury was sustained in an automobile accident. In contrast, of the cases reported since 1951, almost half resulted from automobile accidents, and with only a few exceptions the driver of the car was the victim. There appears to be great liability from the steering wheel as a mechanism for transmitting decelerating force to the upper abdomen. This information is believed to be of importance in car design and in the evaluation of individual patients. The available information concerning the mechanism of injury from Cohn's and the current series of cases is presented in Table 2.

Table 2. Tabulation of Mode of Injury from Two Recent Series
(Cohn et al.[4] and Present Series)

TYPE OF INJURY	NUMBER	PER CENT
Crush	4	7
Fall	14	25
Automobile	25	44
Kick	7	12
Beating	4	7
Not Recorded	3	5
TOTAL	57	100

Of particular interest are two cases (III and VII) in the authors' group of nine patients with traumatic rupture of the duodenum in whom the injury resulted from parental beating of small children, the so-called "battered child syndrome."[18] The orthopedic and neurologic manifestations of such maltreatment of children with attendant high mortality and permanent crippling have been emphasized in the recent literature, but little attention has been given to the severe abdominal injury that may be encountered. It can be noted in Table 1 that auto-

mobile accidents and child beatings accounted for all nine cases of ruptured duodenum in the Denver experience.

Site of the Rupture in the Duodenum (Table 3)

Two-thirds of the injuries occurred in the second and third portions of the duodenum, being evenly distributed between these sections. Both

Table 3. Site of Rupture of Duodenum

(Tabulation from Cohn et al.[4] and Present Series)

LOCATION	NUMBER	PER CENT
First part	4	7
Second part	18	30
Junction of second and third parts	7	11
Third part	17	28
Fourth part	5	8
Not stated	10	16
TOTAL	61	100

the first and fourth parts of the duodenum were involved in a minority of cases, and it is therefore obvious that the entire organ must be carefully scrutinized at laparotomy to be certain that rupture of the duodenum is not responsible when retroperitoneal pathology directs attention to that area. The distribution of sites of injury in this series, as in others, has led to speculation concerning the intimate details of injury.[31, 32] Pressure exerted upon a closed loop has been postulated, the pylorus more or less effectively closing the proximal end and the spine and acute angulation at the ligament of Treitz closing the distal end. Of equal importance may be the relatively fixed location of the duodenum and the consequent explosive effect of transmitted kinetic energy suddenly transferred to static energy in a hollow organ. The whole matter is largely academic, except in understanding and appreciating the damaging effects of relatively minor blows and as an explanation of the occasional case in which no other intra-abdominal injury has occurred.

Sex Incidence (Table 4)

The sex of the patients is predominantly male. This has been a consistent observation in all previous studies. It probably reflects the increased occupational exposure to trauma, the predominantly male participation in contact sports, and, in the current collected cases, the preponderance of male drivers.

Associated Injuries (Table 1)

In earlier reviews on this subject, notably Johnson's[13, 14] and Cohn's,[4] the incidence of associated injuries appears low. Johnson noted that in

Table 4. Sex Incidence

AUTHOR	MALE	FEMALE
Johnson[14]	48	4
Cohn et al.[4]	23	2
Present series	33	4
TOTAL	104	10

nine cases lacerations of the liver were present. Four patients had intraperitoneal ruptures of the intestine, and one a contused pancreas. Cohn listed the more commonly involved organs (liver, spleen, kidney, pancreas, stomach and others) but did not report the incidence of each. This could well be due to lack of data presented by the various authors of the case reports. However, the incidence of associated injuries in the cases gathered from the literature since Cohn's review and in the nine cases we have added appears also to be low (Table 1).

Traumatic pancreatitis was noted as an associated injury in five patients of the present series (Table 1). All of these were due to automobile accidents. Kerry and Glas[19] noted that five of their eight patients with perforation of the duodenum due to blunt trauma had associated traumatic pancreatitis. The significance of this associated injury becomes apparent when it is noted that all five of their patients died. One of the five patients in the present series with associated traumatic pancreatitis also died. Elsewhere in this volume further discussion of this problem can be found.

It is doubtful whether the increased incidence of traumatic pancreatitis can be credited to more alert and thorough exploration of the retroperitoneal area. Perhaps another factor is the increased incidence (Table 2) of automobile accidents and other types of more severe abdominal injuries noted in recent years.

Interval Between Injury and Hospitalization and Definitive Treatment (Table 1)

There was great variation, with survival when therapy was delayed days, weeks or, in one case, even months. Nevertheless, it is probably significant that all of the fatalities occurred in patients in whom the interval between injury and operation was 24 hours or longer. Among the survivors there were six patients in whom retroperitoneal rupture of the duodenum went undiagnosed and more or less untreated for 2½, 5, 6, 14, 18 and 210 days. The fact that this can occur may signify the sterility of duodenal contents in many patients, the localization of contaminating duodenal content by intact peritoneum, and perhaps the tendency to seal intestinal defects by surrounding tissues. In any event,

there seems little question that diagnosis, closure of the rent, and adequate drainage at the earliest possible time are desirable if not imperative.

Preoperative Diagnosis

The preoperative diagnosis of rupture of the duodenum may be exceedingly difficult. The initial symptoms are often mild and physical abnormalities insignificant. After an interval of hours or sometimes days, varying degrees of abdominal, flank and back pain are usually experienced. The pain may be localized at any site from the right lower abdomen to the chest, and on occasion referred pain to the testicle and shoulder has been observed. Nausea, vomiting, hematemesis and shock are sometimes present. Most patients with rupture of the duodenum will have upper abdominal tenderness. Mild to moderate leukocytosis is usually present. Occasionally hematuria is observed, probably secondary to trauma to the right kidney.

Evaluation of roentgenography as an aid to diagnosis of retroperitoneal rupture of the duodenum has been made by Jacobs et al.[11] and by Jacobson and Carter.[12] Several abnormalities may suggest rupture of the duodenum. These are: (1) retroperitoneal air surrounding the kidney and about the crus of the right diaphragm; (2) obliteration of the right psoas muscle and kidney shadows due to extravasation of blood and fluid. In only two of the authors' nine cases were such radiological abnormalities helpful in arriving at a preoperative diagnosis. In the remaining seven cases, the exact diagnosis was not made preoperatively.

Type of Operative Procedure (Table 5; Fig. 1)

A wide variety of operative procedures were used to meet the needs of the particular pathologic condition encountered. The most common method of dealing with linear defects was suture closure after debriding the edges of the wound. This method was safe and highly effective if the retroperitoneal areas were drained. Occasional small fistulas developed, but in all cases these closed without further operative intervention. If a leak from the site of closure occurred, the material invariably found its way to the exterior along the drain tract.

A particular point about the use of drains is necessary. In the authors' opinion, in every case of ruptured duodenum drains should be placed to a point near the closure. The nonperitonealized portion of the duodenum is liable to the same healing deficiencies as the esophagus and the rectum and, despite the most meticulous technique, occasional partial disruption will occur, particularly in the presence of contamination and contusion of tissues. Chemical damage from spilled duodenal content may play a role. For all these reasons, plus the fact that pancreatitis is a common accompaniment of duodenal injury, drainage is necessary. There is no

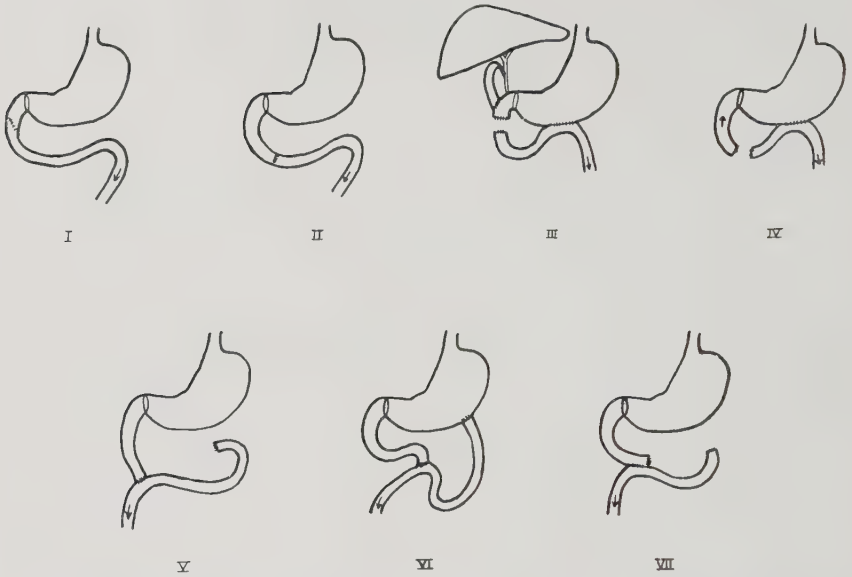

Fig. 1. Diagrammatic representation of various operative procedures used in present series of cases. *I*, Simple closure. *II*, End-to-end duodenoduodenostomy. *III-IV*, Closure of both ends of duodenum and gastroenterostomy. *V*, Closure of distal duodenum and duodenojejunostomy. *VI*, Duodenojejunostomy and gastroduodenostomy. *VII*, Resection of fourth part of duodenum and duodenojejunostomy.

innate liability in a drain, and the dividend may be the patient's life.

In eight of the 37 cases of the present series, the nature of the lesion did not permit simple closure. Circumferential tears were sometimes repaired by end-to-end anastomosis of the duodenum. In other instances, loss of tissue precluded such a maneuver, and usually the jejunum was anastomosed to the proximal duodenum and the distal transected end of the duodenum simply closed. In one case, the fourth part of the duodenum was resected and a side-to-side anastomosis was made between the jejunum and the third part of the duodenum. The most difficult problems were encountered in a few cases in which the tears and disruptions occurred at or very near the ampulla of Vater. Hematoma and ecchymosis may make identification difficult, and the closure may compromise both biliary and pancreatic drainage. One solution to this uncertain situation has been closure of both open ends of the duodenum in conjunction with gastrojejunostomy, cholecystoduodenostomy, and either oversewing of the pancreas or reimplantation of the duct. If the main pancreatic duct is ligated, it would seem advisable to provide retrograde drainage of the pancreas, although this procedure has not been carried out in a patient with rupture of the duodenum at the ampulla of Vater. Other infrequently used techniques are also shown in Figure 1. The various operative procedures that have been used are listed in Table 5.

Table 5. Types of Operations Performed and Mortality in Three Collected Series

TYPE OF OPERATION	JOHNSON[14]		COHN ET AL.[4]		PRESENT SERIES	
	Number of Cases	Mortality (per cent)	Number of Cases	Mortality (per cent)	Number of Cases	Mortality (per cent)
Sutured and drained	24	37	10	10	18	0
Sutured without drainage			4	50	1	100
Sutured and gastroenterostomy	4	75	1	0	0	—
Resection of contused edges and end-to-end anastomosis (either duodenoduodenostomy or duodenojejunostomy)	3	0	2	0	8	12
Drainage only	9	66	2	0	2	50
No operation	8	100	2	50	2	50
Others	4	75	4	25	3	0
Not stated	—	—	—	—	3	66
TOTALS	52	56	25	20	37	16

In an occasional case the presence of duodenal rupture may be difficult to establish at operation. The presence of retroperitoneal hematoma or fluid collection obviously directs attention to the duodenum. Of equal importance are minor degrees of edema, slight bile staining of tissues, and the presence of blood within the stomach without adequate explanation. In the presence of such findings, the duodenum and the head of the pancreas should be mobilized and the posterior surface directly examined. All patients suffering from blunt abdominal trauma should have the stomach and urinary bladder intubated and the contents should be checked for the presence of blood preoperatively. Rectal contents should also be examined. Prior knowledge of such bleeding will sometimes direct attention to otherwise normal-appearing viscera and on occasion avoid catastrophe.

Mortality (Tables 5 and 6)

The mortality from duodenal injury is still high, despite progressive decline over the years. Comparison of the overall death rate in Cohn's series and the current group indicates that little significant reduction has occurred in the past 20 years. The manner in which further improvement in the results of treatment of duodenal injury can be attained is apparent in Table 5. There were six deaths among the collected group of 37. Of these dead patients, one had no drainage, one had no treatment whatsoever, one died of pancreatitis (authors' case) undiagnosed until autopsy and therefore untreated, and one patient had only drainage of a

Table 6. Progressive Decline in Mortality

AUTHOR	TOTAL REPORTED	SURVIVED	MORTALITY RATE (per cent)
Schumacher, 1910	24	2	92
Miller, 1916	22	3	86
Johnson, 1944	52	26	50
Cohn, 1952	25	20	20
Present series, 1962	37	31	16

retroperitoneal accumulation. These four patients could have been salvaged. The circumstances concerning the deaths of the remaining two patients are not given in the published reports. It is apparent that a mortality of 5 per cent or less is attainable, and certainly a conservative estimate of under 10 per cent should be within immediate reach.

One of the principal deterrents to the immediate achievement of this goal is the general viewpoint of the surgical profession concerning blunt abdominal trauma. The need for immediate exploration of all penetrating wounds of the abdomen has been recognized for many years. On the contrary, the indications for exploring the patient with blunt trauma are not the injury itself but rather the catastrophic complications, such as hemorrhage, shock and peritonitis, that may occur late. Particularly with a retroperitoneal organ such as the duodenum, signs of peritonitis should not be awaited, because they may never occur. Bleeding may be insignificant. The situation appears analogous to that which jeopardizes the patient with rupture of the urinary bladder in its retroperitoneal portion. The liability of that lesion and the correct method of management have been appreciated for many years.

SUMMARY AND CONCLUSIONS

This report concerns analyses of reported experiences with retroperitoneal rupture of the duodenum since 1951 and detailed presentation of nine additional cases. In two of these cases, rupture of the duodenum resulted from assaults on children by parents, the so-called "battered child syndrome."[18] The orthopedic and neurologic aspects of this syndrome have been widely publicized, but the occasional severe abdominal injury has received little attention. In contrast to previous collected series, this review of recent experience shows that automobile accidents are the cause of the majority of duodenal injuries, and that impact against the steering wheel is the most frequent mode of injury. The second and third parts of the duodenum are particularly liable to damage, but the other portions of the organ are also ruptured occasionally. Traumatic pancreatitis and fracture of the liver are the most common associated

injuries, but in a high percentage of cases no other intra-abdominal pathology is found. The diagnosis is difficult, and definitive repair is often unnecessarily delayed. A more aggressive approach leading to earlier laparotomy is recommended.

The mortality from rupture of the duodenum remains high (16 per cent in collected series and 11 per cent in authors' cases), and there has been no significant change in the past 10 years as compared to the previous decade. Direct suture of duodenal defects with drainage is the most commonly employed operative procedure, which in this series was without mortality. End-to-end duodenal re-anastomosis and duodenojejunostomy were employed with success in a high percentage of cases. Failure to provide drainage of the retroperitoneal area after repair of this injury is hazardous. The most frequent postoperative complication was the development of duodenal fistulas. There were three such complications in the authors' nine cases, and in the collected series this occurred in 14 per cent of cases. All of the fistulas healed without further operations. Traumatic pancreatitis was the second most common complication and one of serious nature. This condition accounted for the single death in the authors' cases.

It is concluded that present operative methods are adequate and that mortality from this condition can be reduced to very low levels by earlier operation and a more urgent approach to accurate diagnosis and treatment of this as well as other injuries due to blunt abdominal trauma.

APPENDIX

Case I (CGH 181957). A 21 year old man was in an automobile accident. He was in the seat next to the driver. He lost consciousness momentarily. On arrival in the emergency room three hours after injury he was still slightly confused. His main complaint was pain all over the body. Initial examination revealed a small laceration of the forehead, blood pressure 120/70, pulse 92, and respirations 20. There was a linear contusion across the right midabdomen. Vague tenderness with some spasm was localized to the right side of the abdomen. Generalized rebound tenderness was present. Bowel sounds were active. No palpable masses were noted. The hematocrit was 48 per cent and the leukocyte count was 14,900, with a slight shift to the left. The urine was clear, but showed 3+ albumin and 10 red blood cells per high-powered field, on microscopic examination. Serum amylase drawn in the emergency room was 145 Somogyi units. Radiologic examination of the abdomen was normal.

The patient was admitted to the surgical ward and observed closely over the next 8 hours. He developed increased tenderness in the right upper and lower quadrants. The pain in these areas increased in severity and was referred to the right shoulder. No vomiting occurred. The pulse increased to 100. Other vital signs remained within normal limits.

Eleven hours after admission, a laparotomy was performed. There was a small amount of dark brown fluid in the peritoneal cavity. A large retroperitoneal hematoma over the pancreas and in the mesentery of the transverse colon was

noted. Reflection of the hepatic flexure of the colon and the duodenum medially revealed a longitudinal rent in the third part of the duodenum. The pancreas was moderately contused. The ileum 4 inches from the ileocecal junction was also badly contused, as was its mesentery. Closure of the rent in the duodenum plus resection of 12 cm. of ileum was carried out. Drainage of the pancreatic and duodenal area was then effected with multiple large rubber drains through separate stab wounds.

Postoperatively, serum amylase measurements varied from 252 Somogyi units on the first postoperative day to 565 Somogyi units on the seventh postoperative day, with gradual return to normal. Convalescence was uneventful, and the patient was discharged on the eleventh postoperative day.

CASE II (CGH 179850). A 45 year old woman was in an automobile accident in which she was struck in the upper abdomen by the steering wheel. She was immediately taken to a hospital. She complained of difficulty in breathing. After brief observation, she was released from that hospital. She continued to have respiratory distress, and was readmitted 21 hours after injury. No vomiting had occurred and abdominal complaints were minimal. There was moderate tenderness in the upper abdomen. No masses were present. Bowel sounds were absent. Blood pressure was 112/72, pulse 72, and respirations 22. The initial hematocrit was 42 per cent, hemoglobin 13.5, and blood leukocyte count 16,178 with slight shift to the left. Urinalysis was normal.

Roentgenograms of the abdomen were interpreted as normal except for some haziness in the upper right abdomen. The diaphragms were elevated. No free air was noted.

The patient was observed further. Two and one-half days later, the hematocrit dropped to 34 per cent; the pain in the upper abdomen became more severe; and upper abdominal tenderness increased. Eventually rigidity was also present. Vital signs did not change.

Finally, a laparotomy was performed. A small amount of tan fluid was noted in the peritoneal cavity. The mesentery of the transverse colon was discolored, as was the area over the duodenum. The hepatic flexure of the colon was mobilized, and on reflection of the second part of the duodenum medially bile-stained fluid was noted to exude from the retroperitoneal space. A perforation 9 mm. in diameter was found in the second part of the duodenum. There was extensive contusion above and below this. Because of nearness of the biliary and pancreatic ducts, it was elected to place a No. 20 Pezzar catheter in the defect. Drainage of the retroperitoneal area was then effected. Postoperatively the patient did well. Serum amylase on the fourth postoperative day was 95 Somogyi units. On the thirteenth postoperative day, the catheter was removed, and by the eighteenth postoperative day the fistula had healed. The patient was discharged well 15 days later.

CASE III (CH 5587153*). A 5 year old boy was apparently severely beaten by his mother two days before admission. The next day he began to vomit, and this continued to the time of admission.

On admission, the temperature was 103° F., pulse 140, and respirations 28. Severe bruises of the trunk, thighs, arms and face were evident. The abdomen was slightly distended and acutely tender, especially in the right upper and lower quadrants. Rebound tenderness was also elicited. No bowel sounds were heard. The patient appeared emaciated and severely dehydrated.

* We are indebted to Dr. Sidney M. Reckler for permission to report this case.

Roentgenographic examination of the abdomen immediately after admission showed no abnormality except gastric dilatation. Numerous laboratory examinations were normal except for a blood leukocyte count of 20,050 with a marked shift to the left, albuminuria, and 10 to 15 erythrocytes per high-powered field on microscopic examination of the sediment from a centrifuged specimen of urine.

The patient was rehydrated and treated symptomatically, with improvement for 24 hours. The abdomen became soft, and bowel sounds returned. Roentgenograms repeated 2 days later revealed a dilated loop of jejunum in the left upper quadrant. Three and one-half days after admission, the abdomen again became tender and rigid. A laparotomy was performed soon thereafter. No free fluid was found in the peritoneal cavity. A large hematoma surrounded the duodenum. At the ligament of Treitz, there was circumferential disruption involving about half the duodenum. There was also severe contusion of the adjacent 4 inches of jejunum. Resection of the perforated duodenum and jejunum with an end-to-end duodenojejunostomy was carried out. The retroperitoneal space was drained.

Postoperatively, the patient improved slowly. On the seventh postoperative day, the drains were removed and the patient had resumed normal alimentation. A mild case of bronchopneumonia on the fifteenth postoperative day delayed discharge for another 10 days, but following this convalescence was uneventful.

Case IV (CGH 61274). A 45 year old woman was admitted 4 hours after an automobile accident. She complained of a dull persistent midepigastric ache. No nausea or vomiting was present. Her blood pressure was 120/70, pulse 86, and respirations 20. Examination revealed ecchymosis of the periumbilical area and tenderness in the midabdomen. Four-quadrant abdominal paracentesis did not reveal any fluid. No bowel sounds were heard.

The results of initial laboratory examinations included a hematocrit of 37 per cent, blood leukocyte count of 10,800, serum amylase of 268 Somogyi units, slight albuminuria, and 5 to 10 erythrocytes per high-powered field on examination of a centrifuged specimen of urine. Roentgenographic examinations of the abdomen and chest were normal.

A nasogastric tube was inserted, and initially bloody fluid was obtained. After admission, the patient continued with general guarding and rebound tenderness. Twelve hours later, she suddenly went into shock. Abdominal paracentesis produced 5 cc. of bloody fluid. The serum amylase rose to 758 Somogyi units. Shock was corrected, and surgical exploration was then performed. A large retroperitoneal accumulation of blood and bile-stained fluid was present over the pancreas and duodenum. Mobilization of the duodenum revealed a laceration at the junction of the second and third parts of the duodenum, three-quarters of the way about the circumference. Two centimeters of crushed tissue were resected, and a duodenoduodenostomy was performed. The pancreas was noted to be severely contused and fragmented. The debris was removed, and this area was extensively drained.

Postoperatively, the vital signs were stable, but urinary output decreased, and finally, on the second postoperative day, the patient was completely anuric. Fluid therapy was restricted because of this. The patient expired after a brief period of vascular collapse. Autopsy revealed extensive acute necrotizing pancreatitis. The suture line in the duodenum was intact.

Case V (SH 1976*). A 24 year old woman was injured when the car she was

* We are indebted to Dr. James J. DeRoos for permission to report Case V.

driving was struck from behind by another car, throwing her against the steering wheel. She was immediately taken to an emergency hospital, and 3 hours later transferred to another hospital. At that time, she was complaining of pain over the entire abdomen and severe headache. Physical examination revealed abdominal rigidity, absent bowel sounds, and diffuse tenderness. The blood pressure was 100/50. Laboratory examinations were normal except for a blood leukocyte count of 18,000.

Seventeen hours after injury, the patient underwent abdominal exploration. There was bile staining and air in the retroperitoneal tissues. There was considerable fluid in the peritoneal cavity. The fluid about the hepatic flexure of the colon appeared purulent. A rupture of the third portion of the duodenum, surrounded by an abscess, was found. The tear in the duodenum was repaired, and drains were placed into the retroperitoneal area. Multiple transfusions and heavy antibiotic therapy were given postoperatively. Her convalescence was uneventful.

CASE VI (CGH 125173). A 1 year old white girl was admitted to a hospital in an outlying community. Five days before admission, the infant was apparently severely beaten by one of her parents. This information was not available until late in the patient's convalescence. The patient was transferred to Colorado General Hospital 12 hours after her arrival at the outlying hospital. On admission, she appeared moribund. She was acutely dehydrated. The temperature was 98.9° F., pulse 120, respirations 38, and blood pressure 70/0. There were ecchymotic spots over the back and sides of the trunk. Initial examination of the abdomen revealed no masses, and the abdomen was soft. Bowel sounds were absent.

Laboratory examinations revealed the hematocrit to be 27 per cent, blood leukocyte count 11,000, 2+ albuminuria, and the urinary sediment loaded with erythrocytes. Serum electrolytes were: sodium 123 mEq. per liter, potassium 2.4 mEq per liter, chloride 71 mEq. per liter, and carbon dioxide 31 mEq. per liter. Radiologic examination of the abdomen showed the stomach and duodenal loop to be distended with air.

The next 45 hours were required to replace fluid and electrolyte deficits. Abdominal signs of tenderness and involuntary spasm became evident. A laparotomy was performed 2 days after admission. No free fluid was present in the peritoneal cavity. The anterior surface of the duodenum was red and edematous. A retroperitoneal accumulation of foul-smelling fluid was entered at the ligament of Treitz. A complete transection of the third part of the duodenum was identified. The damaged ends were resected and closed. A side-to-side duodenojejunostomy was performed (Fig. 1, VII). The retroperitoneal area was drained.

The postoperative course was prolonged because of the poor general condition of the infant. No serious complications occurred, and the child was discharged 25 days after operation.

CASE VII (CGH 103596). A 12 year old boy fell from a truck, the rear wheel of which passed over his abdomen. Shortly thereafter he was admitted to the hospital in shock. On initial evaluation, the blood pressure was 70 mm. Hg systolic, pulse 144 per minute. The abdomen was tender throughout but maximally in the upper portion. Abdominal paracentesis produced bloody fluid which did not clot. Laboratory examinations were normal except for a hematocrit of 33 per cent and a leukocytosis of 20,300.

Blood transfusions (500 ml.) and plasma expander (250 ml.) improved but did not fully correct blood volume deficiency before operation Laparotomy was

carried out 7 hours after admission. The spleen was macerated, and there was a long linear laceration of the dome of the right lobe of the liver. The remnants of the spleen were removed and the vessels were individually ligated, following which the laceration of the liver was closed with interrupted mattress sutures.

After control of active bleeding, a more leisurely examination of the abdomen revealed bile-stained retroperitoneal tissues about the head of the pancreas. After mobilization of the duodenum and its reflection medially, a longitudinal tear extending from the ampulla of Vater to the third part of the duodenum was exposed. This was closed by direct suture of the edges of the wound, care being taken not to compromise the bile or pancreatic ducts. The right retroperitoneal area and the splenic bed were drained through lateral stab wounds. Three thousand milliliters of blood were given during the operation.

The postoperative course was long and complicated by a duodenal fistula and left subphrenic abscess, which was drained. Both lesions eventually healed, and the patient was discharged 81 days following injury.

Case VIII (St.A.H. 109372*). A 30 year old white man had a seizure while driving and crashed, sustaining a steering-wheel injury to his abdomen. He was immediately taken to a hospital, where he was observed for 36 hours before being transferred. He was complaining of rather severe upper abdominal pain, associated with vomiting of bile-stained material. He passed a bloody stool soon after admission.

Examination revealed the pulse to be 110, respirations 16, blood pressure 130/-80, temperature 99.6° F. The abdomen was scaphoid, with tenderness throughout. Some rigidity was present. The bowel sounds were within normal limits. Initial roentgenographic examinations and laboratory tests were normal. No serum amylase determinations were made. The patient was placed on nasogastric suction and intravenous replacement of fluids and electrolytes. Three days later, the abdominal signs had cleared. However, an upper abdominal mass became evident slightly to the right of the midline. This was thought to represent retroperitoneal hematoma.

Because the patient continued to show signs of upper gastrointestinal obstruction, roentgenographic examination of the upper gastrointestinal tract with barium was carried out, and showed an obstruction at the third part of the duodenum. Eighteen days after the injury, the obstruction remained and an exploratory laparotomy was performed.

A large phlegmonous bile-stained mass was present over the duodenum and pancreas. Bile staining had extended into the transverse mesocolon. This was thought to be due to a rupture of the third part of the duodenum. A side-to-side duodenojejunostomy was carried out between the second part of the duodenum and the jejunum just beyond the ligament of Treitz. Drains were placed to the mass and Morison's pouch.

Bile-stained fluid drained for 10 days postoperatively, and then ceased. The patient was discharged 26 days after surgery.

Case IX (CGH 182643). A 42 year old white man sustained a steering-wheel injury to his abdomen, and was admitted to the hospital 3 hours after the injury complaining of upper abdominal pain. There had been no loss of consciousness. He was vomiting dark brown gastric contents. Examination revealed the blood pressure to be 124/80 and pulse 60. A contused area was present in the mid-epigastrium. Tenderness was marked, with guarding in the upper abdomen.

* We are indebted to Dr. David Barton for permission to include this case.

Bowel sounds were absent. The hematocrit was 42 per cent, blood leukocyte count 8100, and urinalysis normal. Serum amylase was 144 Somogyi units. Radiologic examination of the chest and abdomen was not revealing.

The patient was taken to the operating room 4 hours after admission. A retroperitoneal accumulation of blood was found over the head of the pancreas and duodenum. On reflection of the second part of the duodenum medially, bile-stained fluid was flowing from a small laceration in the third part of the duodenum. This laceration was closed and the area was drained. The pancreas was not badly contused.

Postoperatively, the patient did well. The serum amylase rose to 254 Somogyi units on the third postoperative day, and remained in this range until the twelfth postoperative day. The patient was discharged 19 days after operation.

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* EDITOR'S NOTE: For the sake of completeness, this bibliography contains a number of references not cited in the text. Duodenal perforation secondary to penetrating abdominal injury is an important subject not covered except by these references.

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Liver Trauma

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HISTORICAL

During the nineteenth century, nonoperative treatment was the accepted method of caring for hepatic injuries. The mortality rate was 66.8 per cent.[14] In 1870 Bruns[5] first successfully resected liver tissue and in 1886 Burckhardt[6] controlled acute traumatic liver hemorrhage. The operative therapy of hepatic trauma first became accepted during the early decades of the twentieth century, as improvements in operative technique were perfected.[10, 21, 23, 31, 34] However, there was little or no change in mortality rates until 1941.[3, 9, 17, 36, 39, 42]

During World War II surgeons were able to utilize several general medical advances, in addition to improved surgical technique, to markedly reduce mortality from hepatic injuries for the first time. Paramount among these were blood and plasma expanders, sound knowledge of cardiopulmonary physiology, antibiotics, and improvements in patient transportation. The work of Major G. F. Madding and co-workers[24] is outstanding in this regard. They reported on 829 combat liver injuries with an overall mortality of 27 per cent. After World War II these sound principles of therapy in acute liver injury were applied to civilian surgical practice. Mortality was reduced in some series to 2 per cent and 10 per cent.[18, 41] Other groups[29, 30, 33, 47] were reporting higher mortality figures, however, and the discrepancy between these seemed to be directly related to the type of injury. Thus, Graham[18] reported in 1958 a series of liver injuries primarily caused by penetrating trauma with a mortality rate of 2 per cent. Hellstrom[19] recently reported on 300 cases of nonpenetrating injuries to the liver with a mortality rate of 38 per cent. It is evident that, despite a marked improvement in the care of penetrating injuries, one-third or more of the patients who suffer blunt hepatic trauma and who survive to reach the hospital will eventually succumb to their injuries.

MATERIAL

Ninety-eight patients with acute liver injury were treated at the

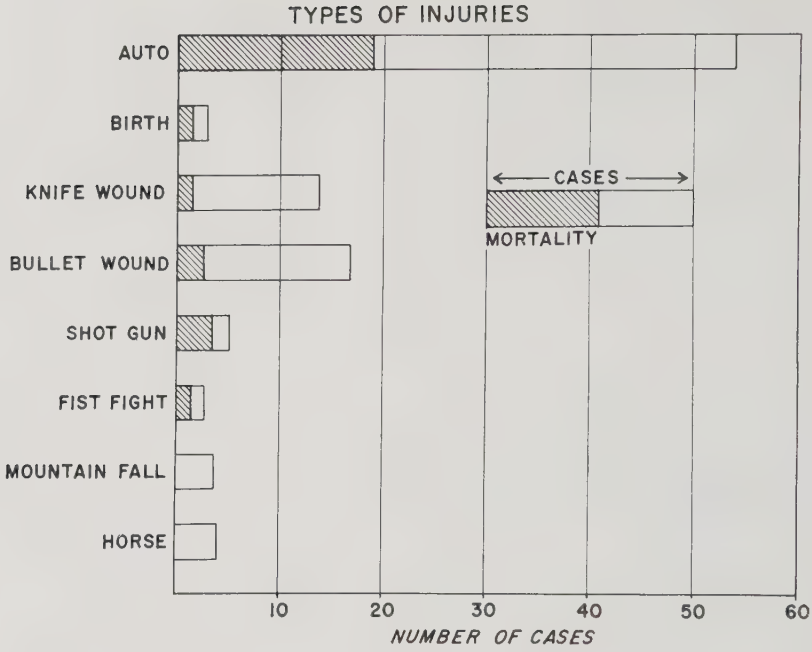
TYPES OF INJURIES
AUTO
BIRTH
KNIFE WOUND
BULLET WOUND
SHOT GUN
FIST FIGHT
MOUNTAIN FALL
HORSE
CASES
MORTALITY
10
20
30
40
50
60
NUMBER OF CASES

Fig. 1

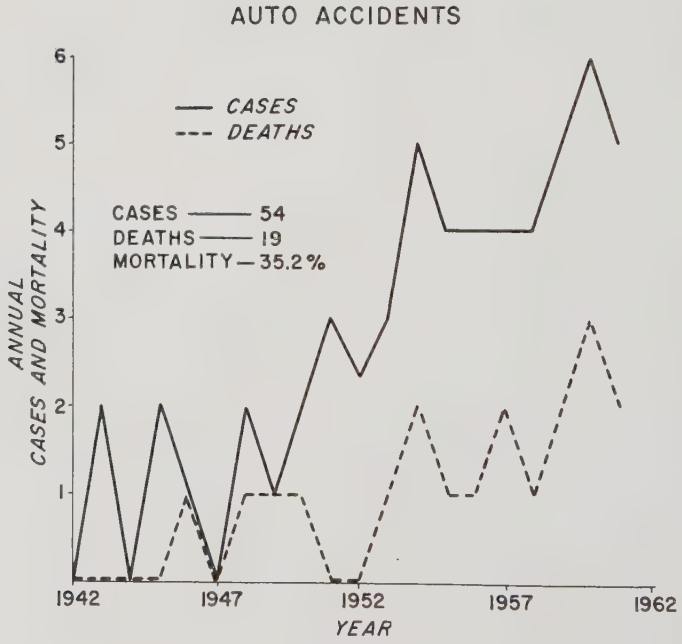
AUTO ACCIDENTS
CASES
DEATHS
CASES —— 54
DEATHS —— 19
MORTALITY — 35.2%
ANNUAL CASES AND MORTALITY
6
5
4
3
2
1
1942
1947
1952
1957
1962
YEAR

Fig. 2

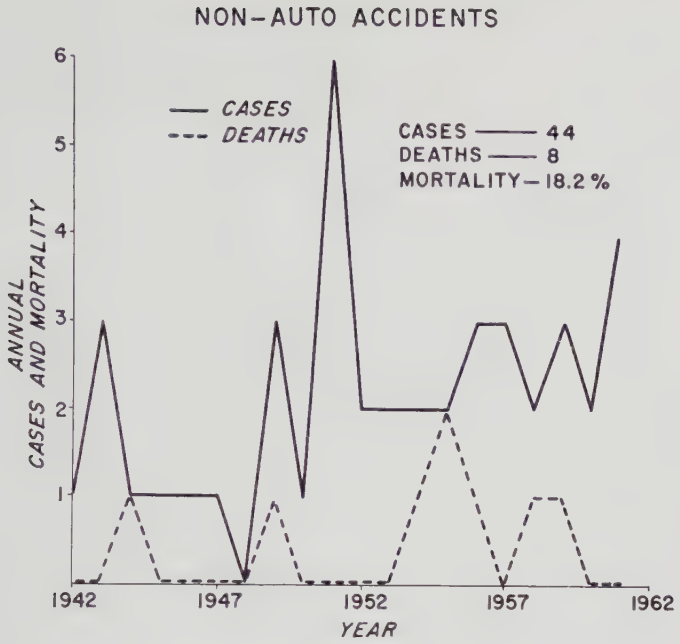

Fig. 3

Colorado General Hospital during the 20-year period ending January 1, 1962. Seventy-one (72 per cent) survived—a relatively low survival rate.

Age. The range was from three days to 73 years, 72 per cent of the patients being between 10 and 40 years old.

Sex. There were 61 males and 37 females.

Location of the Wound. Sixty-eight injuries involved the right lobe only, 12 the left lobe, and 18 both lobes including the hilar structures in five cases.

Types of Injuries and Mortality. Figure 1 lists the types of injuries encountered and gives the mortality figures for each group. In the past few decades automobile accidents have accounted for the majority of the blunt trauma and 55 per cent of our total experience; 35.2 per cent of the patients in this group died (Fig. 2). Non-automobile accidents accounted for 45 per cent of the total experience and most of these were penetrating injuries (Fig. 3). The relatively high mortality rate in this group is augmented by three patients receiving close-range shotgun wounds to the abdomen which were considered surgically incorrectable.

Associated Injuries. Several authors have noted increased mortality when a multiplicity of associated injuries occurs. In our series there was a direct correlation between the number of associated injuries and the mortality rate (Table 1).

Table 1. Mortality in Liver Trauma with Associated Injuries

	CASES	DEATHS	RATE (per cent)
Liver alone	22	—	—
Liver plus one	34	3	8.8
Liver plus two	19	7	36.8
Liver plus three	9	5	55.6
Liver plus four or more	14	12	85.7

DIAGNOSIS

Madding has stated, "The diagnosis of injuries of the liver, when associated with penetrating injuries about the lower chest and upper abdomen, is not difficult. The diagnosis of injuries not associated with penetrating wounds presents a great problem."[26, 27]

Pain referable to the liver injury was the most consistent diagnostic sign. Of 90 patients coming to operation, 74 complained of pain in the right upper quadrant, 39 had pain referred to the right shoulder or scapular area and 64 had generalized abdominal pain immediately before operation. Sixty-seven of the patients were in shock at the time of admission or developed shock in the preoperative period. Shock developing during the operative procedure is not included as a diagnostic sign. This combination of pain referable to the right upper quadrant and hypotension should make abdominal exploration a serious consideration.

Subcapsular bleeding in the liver may produce a palpable mass in the right upper quadrant and will lend support to early exploration. This condition was present in 13 patients in the present series.

Berman[1] reported on the significance of an admission white blood count above 15,000 in the diagnosis of liver injury. Only 41 patients of the present series exhibited white counts in this range; an elevation of the white blood count has not been particularly helpful to us in the diagnosis of liver injury.

The four-quadrant abdominal tap, as popularized by Byrne,[7] was done in 51 patients and was positive in 18. A positive four-quadrant tap is helpful, but negative results may lead to a false sense of security. The four-quadrant tap may be a needless delay if operation must be performed regardless of the findings.

Radiological suggestion of rupture of the liver, as discussed by Schinz,[27] was found in 21 of the 61 cases in which preoperative studies were done. Radiological examinations of the chest, abdomen, skull and long bones are often useful in the patient with multiple injuries, but radiological suggestion of liver injury is rarely the first or strongest diagnostic clue. X-rays should be taken only in those cases in which

they are essential and where there is obviously sufficient time to perform them.

Perhaps the most useful aid in evaluating the patient with possible liver injury in whom the diagnosis is in doubt is careful, frequent repeat examinations. It is imperative during such evaluations to be able to compare findings with those on admission. This can best be accomplished if a single surgeon makes all observations.

PREOPERATIVE CARE

With patients involved in the high-speed crash of an automobile, as in few other areas of surgical experience, diagnosis and preoperative care must of necessity be performed at the same time. If calm reflection on diagnosis is allowed to delay necessary emergency care, the outcome may be fatal.

A thorough but rapid history and physical examination is an immediate necessity in the care of the injured patient. Whether or not shock is manifest, large intravenous routes should be established. For this purpose, 15 gauge needles through which a pint of blood may be infused in one to two minutes are used. Catheters (15 gauge) are probably safer, as they do not tend to become dislodged when the patient is moved. However, the concern is primarily to obtain a large intravenous route, and needles should be used if doing a cut-down will cause significant delay. Blood is obtained at this time for typing and cross matching, and for routine laboratory work. At least two tubes of blood should be sent to the laboratory on admission if there is reason to suspect severe injury and the need for further blood during later operation. Saline or a plasma expander is started immediately in any patient in shock, and in many cases a "central venous catheter" is inserted at this time.[46]

External bleeding is controlled by pressure until the patient reaches the operating room. Elastic bandages are satisfactory for temporary control of hemorrhage but must be removed when the need no longer exists or when definitive care of these injuries is undertaken.

The establishment of adequate pulmonary function is essential to the patient about to have major emergency surgery.[11, 38, 40, 48] A tracheostomy will prove invaluable in many instances and, if serious consideration is given to this procedure, it is probably best performed early in the emergency room. The minutes thus spent may save a life later. The chest wall is stabilized by traction attached to towel clips or other apparatus to prevent paradoxical motion. Sucking chest wounds are sealed with large Vaseline sheets covered with abdominal pads and adhesive tape. Frequent endotracheal suction assures cleansing of the tracheal bronchial tree of previously aspirated blood and vomitus. Oxygen is used liberally.

A nasogastric tube is passed immediately to decompress the stomach. This is left open because the suction machine is too bulky to be easily moved about with the patient.

A Foley catheter is inserted in all severely traumatized patients to assure thorough emptying of the bladder, to yield specimens for urinalysis, to keep the bladder compressed, and to provide a means for measuring hourly urinary outputs of patients in shock after surgery.

Antibiotics in liberal amounts (for example, aqueous penicillin, one million units, and a gram of broad-spectrum antibiotic) are added to the first intravenous bottle and continued postoperatively.

Long bones are quickly splinted, but, if the patient is severely injured, no delay is permitted to obtain films or to assure perfect fracture apposition.

Compatible whole blood is begun as soon as the blood group is determined by the laboratory. If the diagnosis of liver injury is quite certain and the patient is not in shock, operation is undertaken immediately. If shock is present or has developed since admission, it is well to spend a few minutes to determine whether 500 to 1000 cc. of whole blood given quickly will reverse the hypotension. However, operative intervention must not be delayed unduly for many transfusions. A few minutes may be spent giving a liter of blood, but the patient's condition may worsen greatly if surgery is postponed for an hour or two while 2000 to 3000 cc. are transfused. The surgeon must not be deceived by apparent shock conversion after blood transfusion: operative therapy is still necessary.

TREATMENT

Although authors[18] have recently stated that in retrospect they believe liver injuries could have been treated expectantly with a successful outcome, none recommended nonoperative care. In the present series, eight patients were not operated upon, but this was not the treatment of choice. Some of these were admitted in profound shock and expired before an operation could be undertaken. Others lapsed into shock during the early hours of their hospital course and died before reaching the operating room. In this latter group were three patients who might have survived had the principal trauma been recognized earlier. The possibility of hepatic injury in every trauma patient must be considered at the slightest indication and therapy initiated immediately.

Incision

Excellent exposure is mandatory for the careful repair of hepatic injuries and possible associated intra-abdominal trauma. Generally, abdominal incisions, either vertical or right subcostal, offer adequate exposure to the traumatized liver. However, it is occasionally necessary

to extend these incisions across the right costal margin and into the chest to afford better visualization.

Exploration

Rapid assessment of the intra-abdominal pathologic state should be accomplished before attention is directed to the liver injury. Major nonhepatic vascular hemorrhage will require priority attention but other organ injuries should be assessed and cared for following repair of the hepatic wound. Fragmented tissue and blood clots are best removed by the cupped hand and liquid blood by the use of many moist laparotomy pads in the manner described by Madding.[26] The usual suction apparatus is almost useless for this procedure. Adequate debridement of nonviable or questionable hepatic tissue is the first essential in the care of the injured liver. It is preferable to excise some viable tissue rather than to leave potentially necrotic liver within the peritoneal cavity.

Although it has been stated that very few liver injuries will still be bleeding at the time of laparotomy, in this series 40 per cent of the patients involved in automobile accidents had active brisk hepatic hemorrhage. At this point, assessment of the severity of the liver injury is necessary. The majority will consist of linear or fragmented lacerations involving the dome of one lobe and extending for a variable distance into the substance. These, for the most part, can be repaired by simple techniques to be discussed. However, in 14 cases in this series the operative description of the liver damage suggested that subtotal hepatectomy was the procedure of choice. No hepatectomies were done, however, and 11 of this group expired. Subtotal hepatectomy has been recommended as a safe procedure without loss of hepatic function[32, 35] and should probably receive more serious consideration in the future.

Where feasible, individual ligature of vessels and bile ducts accomplishes the most satisfactory control of bleeding from damaged liver and avoids large areas of nonviable tissue caused by mass ligature with mattress sutures.[23, 25, 27, 41] If bleeding is so massive that exposure of vessels and bile ducts for individual ligature is impossible, a temporary hemostatic pack or hand control of the vascular supply of the liver should be considered. Once bile stasis and hemostasis are obtained, it is usually unnecessary to reconstitute hepatic architecture. Frequently, the large mattress sutures used for this purpose place the encompassed tissue in vascular jeopardy.

The use of absorbable hemostatic material has been condemned if hemostasis can be obtained by more reliable means such as ligatures.[8, 12, 16, 22, 43] However, reconstituted methylcellulose (Surgicel) is most effective in controlling hemorrhage from an hepatic surface such as that seen beneath a subcapsular hematoma. In the present series absorbable hemostatic material was used in 33 cases and no proven dele-

terious effects have resulted from its use. Madding[24] was the first to condemn the use of a pack left within the abdomen for hemostasis and supported his vigorous condemnation with statistics. Laparotomy pads or vaginal packing were used in 20 patients in this series but in the past ten years have been used in only five. This method of hemostasis serves only to wall off a pool of blood and bile and does not provide either intravascular control or adequate external drainage.

The essential role of adequate perihepatic drainage has been demonstrated in the past[24] but in our series would have to be considered inadequate in many cases (Table 2). Large flexible soft-rubber drains of

Table 2. Drains Used in Liver Surgery

DRAINS	CASES
0	10
1	50
2	20
3	9
4 or more	1

adequate number should be placed to all points of the hepatic injury and brought out through separate stab wounds. Drainage through the primary abdominal or thoracic incision is probably to be condemned.

Following care of the liver injury, meticulous examination of the remainder of the peritoneal cavity should be undertaken. Particular attention should be paid to areas where hidden injury may occur. This would include the extrahepatic bile ducts, retroperitoneal duodenum, pancreas and major vessels, and the bladder.

COMPLICATIONS

Complications in this series include all untoward or unexpected occurrences within 30 days of surgery, as summarized in Table 3. The high incidence of complications is related to the type of injury and is similar to that seen in other series.[41] It is difficult to reduce this high complication rate, but certain factors seem applicable to future patient care. Postoperative infections will be reduced only when extensive debridement, adequate drainage, better hemostasis and more meticulous attention to associated injuries are routinely achieved.

Of the nine postoperative biliary fistulas, seven closed spontaneously within four weeks, reaffirming the value of conservative therapeutic approach. Two fistulas were successfully closed at reoperation. At the original operation attention should be directed toward any major bile

Table 3. Complications

Infections (Total patients infected, 32)		
Wound	19	
Intrahepatic	5	
Subphrenic	14	
Intraperitoneal	7	
Pleural	4	
Biliary fistula	9	(7 temporary)
Wound dehiscence	3	
Renal shutdown	5	(possibly 2 others)
Bleeding diathesis	9	
Myocardial infarction	6	
Cerebrovascular accident	2	
Thrombophlebitis	5	
Liver emboli	—	
Pulmonary emboli	4	
Pneumonia	13	

duct injury, and either a primary repair or closure over a T-tube should be instituted in most cases.

Acute tubular nephrosis[13, 15, 28] occurred in seven patients, secondary to shock and large volume transfusions. The prophylaxis of shock therapy and careful use of matched blood is well known. Physicians responsible for patient hemodialysis will be able to cope with the problem of "renal shutdown" more effectively if they are quickly informed of this complication. The use of an indwelling urinary catheter is invaluable in making the diagnosis.

The hepatorenal syndrome was described in detail by Hayd[20] and Boise,[2] but the actual existence of the syndrome is now open to serious question. Some writers believe it is seen only in cases of pre-existing cirrhosis and possibly does not play a significant role in acute trauma. No clearcut cases were seen in our series.

Bleeding diatheses occur more frequently as the magnitude of blood transfusions and operative procedures increases; 13 such cases were observed in this series, and six of the patients expired while still bleeding. When excessive uncontrolled hemorrhage occurs, whether at operation or in the early postoperative course, immediate consultation with the Hematology Service should be obtained. Laboratory work is of limited value in the diagnosis at present, but considerable work is being done to explain the bleeding diathesis. Prothrombin time, clotting time, clot retraction time, clot lysis and platelet counts are done with the hope that they may occasionally provide assistance. Specific therapeutic measures such as blood in silicon bags, fibrinogen, calcium gluconate, polybrene, antihistamines, ACTH, vitamin K and epsilon aminocaproic acid have all been used successfully at the Colorado General Hospital.[3]

The usual measures for the prevention of wound dehiscence, embolic phenomena and pneumonia should be utilized.

PREVENTION[44, 45]

Extensive research on safety factors for the automobile passenger was undertaken several years ago. Safety glass, safety door locks, and improved headlights, brakes and visual clearance resulted, and these have become standard equipment on most vehicles. Other factors such as seat belts, reverse seats, head rests, safety dashboards and safety steering wheels have not become so well established. Surveys have proved that these changes will reduce the seriousness of accidents. The public must be educated to demand and use these devices for their own protection; such education is partly the responsibility of the medical profession.

SUMMARY

The mortality from acute traumatic liver injury has been reduced from 66 per cent to approximately 30 per cent over the past two decades. The mortality today is largely from blunt, nonpenetrating injuries, the preponderance of which are caused by automobile accidents. Advances in knowledge of surgical physiology and improvements in surgical technique have been largely responsible for the better survival today. Further improvements will ensue from the prevention of accidents, earlier diagnosis and operation, and the prevention of serious and frequently fatal complications.

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Ruptured Spleen

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High-speed automobile travel and frequent road accidents, sports injuries and industrial mishaps result in an increasing incidence of traumatic rupture of the spleen. This is reflected in the numerous recent publications on the subject. According to Maughon,[3] the ratio of admissions for splenic trauma to general surgical admissions increased approximately threefold between 1951 and 1959. Despite familiarity with the clinical features of the condition, clear recognition of the serious sequelae of improper management, and the aid of modern medical facilities and techniques, the mortality remains high. There is a corresponding excessive morbidity. In the present series, the mortality and morbidity rates were 28 per cent and 64 per cent respectively.

It is possible that these results reflect the nature of the injuries sustained and therefore represent maximal salvage rates and optimal utilization of current knowledge and techniques. Obversely, it seems probable that current diagnostic methods could be more fully utilized and results improved by definition of the inadequacies in diagnosis and management as presently practiced. This analysis of the experience of the surgical service of the Colorado General Hospital during the past ten years was directed to this purpose. Particular attention was devoted to determination of correlations between outcome and extent and mode of injury, the interval between injury and treatment, and an evaluation of diagnostic methods. Detailed study has been devoted to the records of the 14 patients who died.

MATERIAL

Traumatic rupture of the spleen occurred in 50 patients. Seventy-two per cent of these resulted from blunt abdominal trauma, 10 per cent from penetrating wounds (gunshot and stabbing). In nine cases (18 per cent) the splenic rupture occurred during surgical operations or diagnostic

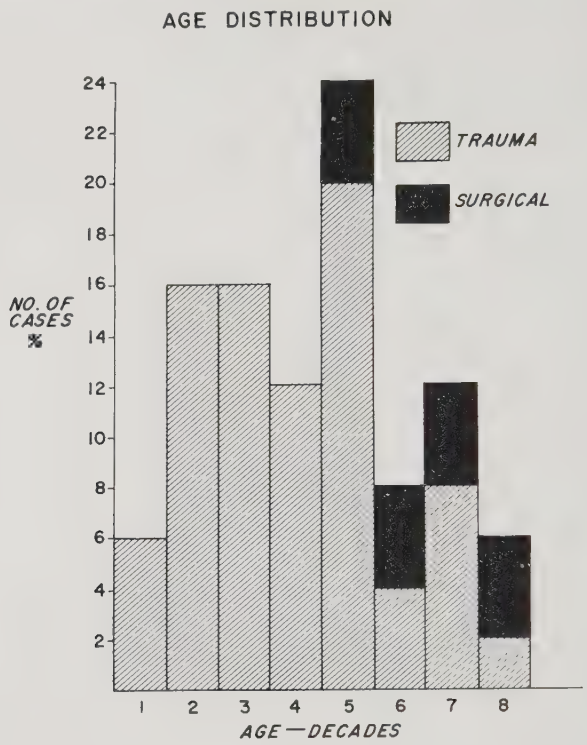

Fig. 1

aspirations of the spleen. From a statistical point of view, surgeons were more dangerous than bullets or bandits, and roughly one-fourth as dangerous as automobiles. The age distribution is shown in Figure 1. The occurrence of iatrogenic splenic trauma only in the older age group probably reflects the age distribution of the ward population in this hospital.

CLINICAL RESULTS

There were 11 deaths in the hospital, and three additional patients died later of bowel complications. In one case, a fecal fistula and subphrenic abscess developed two months after injury. Another patient died of complications of duodenal ulcer surgery including splenectomy. A third patient died of intestinal obstruction which developed 40 days after splenectomy. The fatal cases are listed in Table 1, along with the causes of death and other data. Infection was responsible in seven cases. Only one patient died of shock. Nine of the deaths (64 per cent) occurred in patients in the sixth, seventh and eighth decades. Thirteen of the 14 deaths (93 per cent) occurred in patients over 50 years of age. The other

Table 1. Causes of Death

CAUSE	TYPE OF INJURY	SURVIVAL TIME	OPERATION	AGE IN DECADES
Pneumonia	Splenic biopsy	10 days	Splenectomy	8
Pneumonia and renal tubular necrosis	Splenic biopsy	9 days	Splenectomy	7
Shock	Gunshot—bowel and liver	1 day	Splenectomy	7
Pneumonia	Surgical trauma	11 days	Splenectomy and other surgery	8
Intestinal obstruction and perforation	Surgical trauma	40 days	Splenectomy and other surgery	6
Infection	Blunt trauma—liver, pancreas	19 days	Splenectomy and other surgery	5
Hepatic laceration	Blunt trauma—also head injury	1 day	Laparotomy—death in operating room	6
Hepatic laceration	Blunt trauma—also renal injury	1 day	Splenectomy and partial hepatectomy—death in operating room	3
Pneumonia	Blunt trauma—also bowel and bladder injury	9 days	Splenectomy and genito-urinary surgery	8
Cardiac arrest (at operation)	Penetrating wound—pancreas, bowel	1 day	Splenectomy and other surgery	5
Subphrenic abscess and fecal fistula	Blunt trauma—pancreas, kidney, bowel	2 months	Splenectomy and other surgery	6
Peritonitis; duodenal fistula; dehiscence	Surgical trauma	8 days	Splenectomy and other surgery	6
Cervical spinal cord compression	Blunt trauma—kidney, head and neck	10 days	Splenectomy	5
Multiple injuries	Blunt trauma—pancreas, kidney, bowel, head	2 days	None	5

patient, aged 30, died of multiple injuries including rupture of liver and kidney as well as spleen. Five of the 14 deaths (36 per cent) occurred in patients whose splenic injury was iatrogenic. If one excludes these cases, the mortality from traumatic rupture of the spleen was 22 per cent.

Complications occurred in 64 per cent of the patients. These were evenly divided between those occurring within the immediate period after injury and operation (first 48 hours) and those occurring later. Atelectasis, pulmonary infection, intra-abdominal and wound infections were the principal types of complicating conditions occurring post-operatively.

FURTHER ANALYSIS

In an attempt to define and clarify the reasons for a high mortality and complication rate, numerous possible etiologic factors have been examined, among which are severity of trauma as judged by incidence of associated injuries, intervals between injury and admission to the hospital and operation, size of the wound in the spleen, age of patient, and mechanism of wounding.

Severity of injury obviously has a bearing upon the outcome. It will be noted in Tables 1 and 2 that nine of the 14 deaths occurred in patients

Table 2. Influence of Multiple Injuries on Complication and Mortality Rates

	NUMBER OF CASES	COMPLICATIONS		DEATHS	
		Cases*	Per cent	Cases	Per cent
Spleen only	16	12	75	5†	31
Spleen + 1 other organ	9	7	78	0	0
Spleen + 2 other organs	6	2	33	1	17
Spleen + 3 other organs	19	13	68	8	42
TOTALS	50	34	64	14	28

* Includes fatal cases.

† All 5 cases due to iatrogenic splenic rupture during operation for upper gastrointestinal hemorrhage (3 cases) and as a result of needle puncture of spleen (2 cases).

with multiple organ injuries. Two other patients died after splenic biopsy. Both suffered from blood dyscrasias and general debility, for which they were hospitalized at the time of the injury. The remaining three deaths occurred in patients undergoing emergency gastric surgery for bleeding. None of these deaths was directly related to the splenic tears except for the general influence of mild to moderate hemorrhage and extension and prolongation of surgical operations. In other words, none of the deaths could be attributed to rupture of the spleen alone. All patients in whom rupture of a normal spleen was the only injury recovered. The incidence of injury to other viscera is shown in Table 3. There appears to be a direct correlation between the number of organs

Table 3. Associated Injuries After Blunt Trauma

SITE	PERCENTAGE
Head	31
Bowel	21
Liver	19
Kidney	19
Pancreas	17
Ribs (fractures)	50

injured and the mortality, ranging between zero in those with injury to the spleen only or to the spleen and one other organ (including fractures) to 17 per cent in those with injuries to the spleen and two other organs, and finally 42 per cent in those patients with rupture of the spleen and damage to three other organs (Table 2). Such correlations simply emphasize the obvious. Of more interest is the very high complication rate in all groups of patients. Our conclusion from this is that the more severely injured patient is less able to withstand the occurrence of complications, and that any improvement in the management of these patients will be brought about by better control and resolution of life-endangering complications. The facts brought out by this analysis are all consonant with those in the next section below, showing that the age of injured patients is an important determinant of the outcome after injury.

Age distribution of patients is shown in Figure 1. As pointed out above, 93 per cent of the fatalities occurred in patients over 50 years of age.

The interval between wounding and transfer to the hospital, and the interval between arrival at the hospital and operation could be accurately established in only 27 cases. The average interval between the accident and hospital admission was between one hour and two hours, the shortest interval being 30 minutes and the longest 33 days. The interval between admission and operation averaged between two hours and three hours. The shortest time was 50 minutes and the longest 38 days, in a patient with delayed rupture of a subcapsular hematoma. These data were studied extensively, but no correlation could be established between the complication and mortality rates and the intervals between injury and definitive control of hemorrhage. It does appear that moderate delay during establishment of the diagnosis, preparation for operation, blood volume replacement and emergency care of other injuries does not prejudice the patient's chances of recovery or greatly increase the severity or incidence of complications.

The type of wound in the spleen was described in 45 of the 50 patients. No correlation could be established on the basis of magnitude of injury to the spleen. There were numerous instances in which small wounds bled freely and other examples of complete avulsion or maceration of the organ with no more than ordinary blood loss. Fibrinolysis was not recognized. When bleeding continued after operation, it was due to other injuries except in two instances in which inadequate hemostasis in the splenic pedicle necessitated reoperation.

CLINICAL EVALUATION

The diagnosis of splenic rupture can be difficult, and the decisions regarding management hinge on a correct and rapid diagnosis. If oper-

ation is not undertaken in the presence of splenic rupture, the mortality has been quoted in the range of 90 per cent.[4] On the other hand, if the wrong diagnosis is made, a patient who may have other serious injuries is subjected to a needless operation with prejudicial influence on chances of recovery.

In temperate zones, the occurrence of spontaneous rupture of the spleen is rare. Consequently a history of trauma will usually be found, although it may be remote in time or possibly mild in degree. In this series, the most frequent mode of injury was blunt trauma to the upper abdomen and lower left chest, inflicted by automobile steering wheels in the case of the driver, and by impact after free movement in the vehicle cab in the case of the passengers. The severity of injuries involved is not surprising when the magnitude of the forces involved is considered, i.e., deceleration values of the order of 75 g. at 47 miles per hour, involving a loading as high as 7500 pounds p.s.i. pressure on the thorax by the steering column.[5] It is not surprising that head injuries are a common accompaniment of abdominal trauma and constitute the most frequent associated injury except for fractured ribs (Table 3).

By far the most constant physical finding is that of abdominal pain. This was present in 95 per cent of the victims of splenic rupture due to

Table 4. Location and Onset of Pain

	PERCENTAGE OF OCCURRENCE
Abdominal	95
Left upper quadrant	55
Right upper quadrant	15
Generalized	35
Kehr's sign	7.5
Delayed until after admission	15

blunt trauma. The distribution is seen in Table 4, and it is interesting to note that Kehr's sign (pain referred to the left shoulder) was relatively uncommon. The left upper quadrant is the most frequent location of pain.

Signs of peritoneal irritation (guarding, muscle spasm, rigidity and rebound tenderness) caused by intraperitoneal hemorrhage are usually found on examination, and on occasion may be accompanied by increasing abdominal distention. Abnormal signs may not be found during examination soon after injury, and in several cases in this series the nature of the injury was not obvious for several hours after the accident and initial examination. Repeated abdominal examination must therefore be made in the doubtful case.

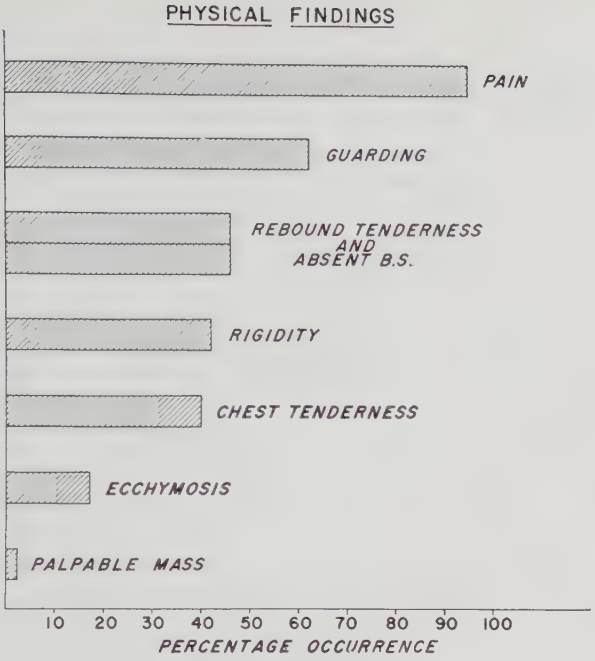

Fig. 2

A summary of the frequency of abnormal physical findings in the present group of cases is given in Figure 2.

Serial measurement of vital signs is an important adjunct to the history and physical examination. Gradual decline in blood pressure and rises in pulse and temperature were commonly observed, as listed in Table 5.

Table 5. Cardiovascular Dynamics and Temperature

PARAMETER	PERCENTAGE
Blood pressure falling	62
Pulse rate rising	60
Temperature rising	20

In 54 per cent, the impression of clinical shock was made on the basis of such findings, accompanied by restlessness, pallor, low skin temperature, and diaphoresis. It is noteworthy that in 40 per cent of the patients the pulse and blood pressure were within normal range.

Diagnostic Aids

After completion of the clinical examination, the examiner may be left with doubt as to the extent of injury and as to whether surgical

intervention is necessary. Patients with head injuries may pose particularly difficult problems. In automobile casualties, head injury was one of the most frequent injuries associated with splenic rupture (Table 3). Many of these patients sustained clouding of consciousness, and 32 per cent of the patients injured in automobile accidents had lost consciousness at some time subsequent to the accident.

The diagnostic aids which were routine when circumstances permitted were: (1) laboratory studies, (2) roentgenographic examination and (3) abdominal paracentesis.

LABORATORY STUDIES. A fall in hematocrit was observed in 62 per cent of the cases. The white blood cell count was recorded in 30 cases. There was an elevation above 10,000 leukocytes per cubic millimeter in 80 per cent of the patients in whom counts were obtained, and a shift to the left in the differential count in 54 per cent.

ROENTGENOGRAMS. Contrary to findings in the other series,[2] x-ray examinations were found to be of limited value in diagnosis of splenic rupture. Generalized signs of intra-abdominal hemorrhage, such as haziness or obliteration of the psoas outline, were recorded in 11 per cent. But in only one case were localized signs (increase of splenic shadow and indentation of the greater curvature of the stomach) leading to a radiographic diagnosis of splenic rupture recorded. It is worth noting that in 50 per cent of splenic lacerations due to blunt trauma, the ribs overlying the spleen were found to be fractured on x-ray examination. Figure 3 shows the incidence of positive x-ray findings.

ABDOMINAL PARACENTESIS. This was the single most constant aid to diagnosis. It was positive in 84 per cent of the patients in this series. This parallels very closely the findings of others.[1, 4] It is important that all quadrants of the abdomen be aspirated, for the distribution of positive taps is fairly evenly distributed, with, however, slightly higher incidence in the left lower quadrant. Some confusion can occur if the needle is passed deep into the peritoneal cavity and a retroperitoneal hematoma is entered. This should be remembered particularly in tapping the lower abdomen in the presence of pelvic fractures. Care should be taken in choosing the site of insertion to avoid the superior and inferior epigastric vessels and the liver and spleen. The direction of the needle is toward the paracolic gutters. When there is free blood in the peritoneal cavity, it can usually be aspirated immediately after perforating the peritoneum. Aspirated blood does not clot if the source of the aspirate is free blood in the peritoneal cavity.

Finally, emphasis should be placed on the importance of repeated evaluation of the patient, with continuous recording of vital signs and frequent re-evaluation of evidence for progression or remission. In this series, the final decision to operate was made on the basis of clinical

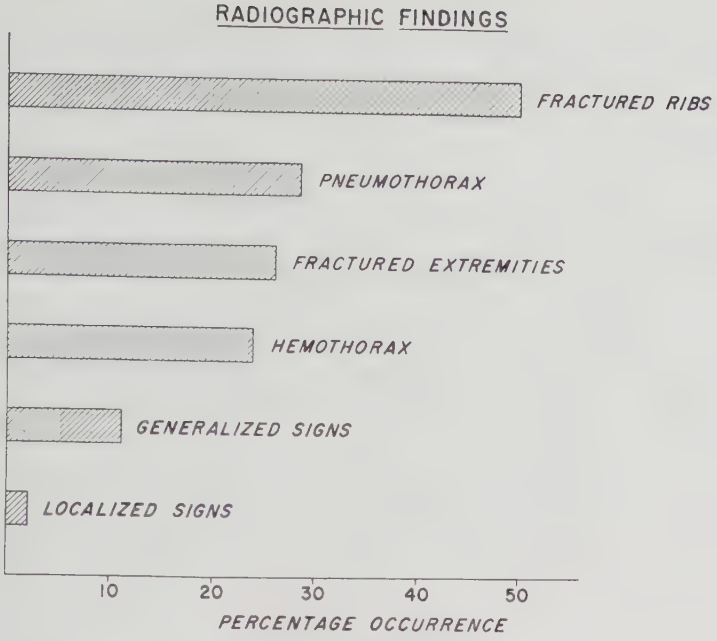

Fig. 3

deterioration in 89 per cent, lack of improvement in 16 per cent, and deterioration of laboratory status in 60 per cent of the cases.

TREATMENT

The treatment of splenic rupture is excision. In this series all but two of the patients were treated by splenectomy, and in these two cases death occurred before or shortly after the patients reached the operating room.

The approach in 92 per cent of cases was made via a vertical paramedian laparotomy incision. Splenectomy was performed with individual ligation of the vessels of the splenic pedicle when possible (76 per cent). Associated intra-abdominal injuries required further surgery in addition to splenectomy in 44 per cent of the cases.

Postoperative Course and Management

Postoperative complications occurred in 64 per cent of these patients. Infection was by far the most frequent complication, and in most instances the site of infection was the lung. This is not surprising in view of the fact that half of the patients had fractured ribs and others had rather extensive chest wall damage without fractures. The serious import of pulmonary sepsis complicating splenic injury is demonstrated by the

fact that four of the 14 deaths resulted from pneumonia. Other infections included six wound infections and five intra-abdominal infections. In some of these patients there was heavy bacterial contamination due to bowel penetration. There was a wide variety of less common complications, including unexplained fever, hemorrhage, transfusion reaction, abortion, and cardiac arrest at laparotomy.

By far the most promising area for improvement of overall results in this group of patients is in prevention and control of pulmonary complications. Without elaborating details, it can be said that vigorous efforts to avoid and clear atelectasis and promote tracheobronchial cleansing should be made. Bronchoscopy and tracheobronchial suctioning were insufficiently utilized, and tracheostomy was too often performed as a last desperate measure. Heavy antibiotic therapy appropriately controlled by bacteriologic study is indicated in many if not all cases of chest and abdominal trauma sufficient to rupture the spleen. These suggestions and remarks are obviously more pertinent to some cases than to others. The elderly and debilitated are at much greater risk, and they in particular require the utmost in detailed care to survive.

DISCUSSION

In order to evaluate the effectiveness of current methods of management of splenic rupture, the experience during the past 10 years at the Colorado General Hospital has been reviewed. Many interesting facts have appeared from this study, among which are the high morbidity and mortality (64 per cent and 28 per cent respectively). In seeking an explanation for this, a number of possible factors were examined. The magnitude of the injuries sustained, as indicated by the crude criteria of the number of organs damaged, appears to be directly related to the mortality. The age of the patients is another factor which has a strong influence on the final outcome. All deaths but one occurred in patients over 50 years of age. Infection was by far the most common postoperative problem, being approximately seven times more frequent than any other complication. Infections of the respiratory tract were twice as common as those occurring in the abdomen or in the wound. Infection within the abdomen of chest appears equally serious, as judged by the number of fatalities ascribable to each.

A number of negative correlations have appeared, all of which were or could have been anticipated. (1) There was no relationship between the interval between wounding and the success rate of ultimate control of hemorrhage. This in no way detracts from the long-sought objective of early surgical treatment and restoration of homeostasis. It does mean, however, that brief pauses for diagnostic maneuvers, procurement of blood, resuscitation, and treatment of other injuries, do not imply mis-

management. (2) There was no relationship between the type of splenic wound and the outcome. (3) There was no correlation between survival and the type of injury, i.e., blunt vs. penetrating. (4) Finally, there was no correlation between the incidence of complications and the magnitude of trauma. All groups had a high complication rate, but the complications were more serious in the highly fatal multiple injury group. It seems apparent that future improvement in the care of these patients will come from better management of post-injury and postoperative complications. Of particular importance will be prevention and control of respiratory complications, among which infection is predominant.

Evaluation of diagnostic methods including clinical appraisal, laboratory aids, roentgenography, and observation over a short period of time has given indication of the adequacy of current approaches to this problem. The degree of reliance which one can place upon various pieces of information has been estimated. It is apparent that no single observation can be utilized to the exclusion of others, and that proper synthesis of data allows the correct diagnosis in virtually 100 per cent of the cases.

SUMMARY

Review of recent experiences in the management of patients with ruptured spleen has revealed high morbidity and mortality rates. The latter can be related to the magnitude of the injuries and the ages of the patients. A surprising incidence of iatrogenic splenic injury with fatal outcome was found. The most common complications were infection in the respiratory tract, intra-abdominal infections, and wound infections. It is concluded that further improvement in management of this group of patients will come from more effective control of respiratory complications and infection. Present diagnostic methods appear adequate.

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The Management of Colon Injuries

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THE PROPER MANAGEMENT of colon injuries has been debated for many years. At the conclusion of World War I, primary closure of colon injuries was regarded as the treatment of choice.[11, 37] At the conclusion of World War II, most surgeons felt that exteriorization or colostomy was the preferred form of therapy.[26, 36] On the basis of this experience, routine exteriorization of all colon injuries has been advocated in many medical centers since the early 1940's. In 1951, the first post-war article to emphasize primary closure without colostomy was published by Woodhall and Ochsner.[39] Since then other authors have reported such favorable experiences with primary closure[18, 35] that the pendulum is now swinging back toward this method of therapy.

Is there a fundamental difference between war injuries and civilian injuries? Is there justification for treating these in different ways? This article will attempt to present the data necessary to answer these questions.

MORTALITY

The overall mortality rate from colon injuries has steadily fallen from 90 per cent during the Civil and Spanish-American Wars, to 58 per cent in World War I, 37 per cent in World War II, and 15 per cent in the Korean War. The improved statistics are due to a variety of factors, particularly blood transfusions, but also plasma, antibiotics, adoption of the policy of routine exploration of all penetrating abdominal wounds, rapid evacuation and earlier treatment.

Most deaths in patients with colon injuries have not been due to the colon injury per se, but rather to associated injuries, as to the liver, spleen, pancreas, kidneys, aorta or vena cava. The mortality rate for injuries involving the colon alone was 11 per cent in 237 patients during World War II, and was 0 in 71 patients in the post-war era (Table 1). In contrast, the mortality rate was 61 per cent in 353 soldiers with other associated injuries, and 14 per cent in 240 civilians with multiple injuries. These figures support the opinion that colon injuries alone are usually not fatal. They also suggest that there are differences between military

Table 1. A Comparison of Mortality Rates from Simple Colon Injuries and Multiple Injuries

	AUTHOR	YEAR	SIMPLE COLON INJURIES No. Pts.	SIMPLE COLON INJURIES Percentage of Deaths	MULTIPLE INJURIES No. Pts.	MULTIPLE INJURIES Percentage of Deaths
World War II	Porritt[31]	1946	177	11	263	69
	Taylor[36]	1948	28	18	39	31
	Imes[17]	1945	32	6	51	43
	TOTAL (1940–1945)		237	11%	353	61%
Civilian Experience	Isaacson[18]	1961	28	0	100	13
	Roof[35]	1960	43	0	140	15.7
	TOTAL (1945–1959)		71	0%	240	14%

and civilian injuries. Military wounds are frequently produced by high-velocity missiles which result in extensive tissue damage in a wide area about the missile's path. In contrast, most of the penetrating wounds seen in civilian practice are due to knives or standard missiles which usually do not exhibit such extensive damage.

The time lag from injury until treatment rarely exceeds a few hours in civilian injuries, while treatment of military wounds may be delayed for much longer periods of time. The simple colon perforation that produces few complications when closed early can result in fatal peritonitis if treatment is delayed too long. The facilities and personnel necessary for close postoperative supervision are often superior in civilian hospitals as compared to front line hospitals. Postoperative complications that can easily be managed in well staffed hospitals become major problems if they develop when the patient is being transferred from a front line hospital to a larger base hospital. During World War II, Ogilvie observed how poorly patients with abdominal injuries tolerated being moved postoperatively.[26]

INDICATIONS AND TIMING OF OPERATION

As a rule, any patient with a penetrating abdominal wound should undergo exploratory celiotomy. Even though no clinical signs of visceral injury are evident, it is generally felt that the risk of not operating on a damaged viscus is far greater than the risk of a negative abdominal exploration.

In caring for the patient with multiple injuries, it is important to bear in mind the priority for treating these. Celiotomy should not be performed until (1) an obstructed airway, (2) bleeding outside the abdominal cavity, (3) shock, (4) head injuries and (5) chest injuries have been corrected or stabilized.

Patients in shock who fail to respond to blood and fluid therapy may be bleeding from intra-abdominal injuries and should therefore be explored in spite of their low blood pressures.

Although common sense dictates that patients with peritonitis from intra-abdominal injuries should be operated upon as soon as possible, Chunn[5] and others[17, 19, 26, 36] have shown statistically that a few hours delay is of little significance. The time spent in preparing the patient for operation with blood and plasma is, therefore, time well spent.

CHOICE OF OPERATION

The choice of operation in any given case is determined by several factors: the location and extent of the colon injury; the mode of injury; the general condition of the patient; the experience of the surgeon; and the facilities available for postoperative care.

1. Primary Closure Without Colostomy

This is the procedure of choice for small wounds whose margins bleed freely. During World War I, Fraser[11] advocated this method of treatment for all colon injuries except where the damage was extensive.

During World War II, many surgeons found that exteriorization was indicated so frequently that a policy of routine exteriorization was established in several hospitals. The explanation for this change in therapy between wars probably lies in the types of wounds treated. Many of the severely injured patients who could never have been operated upon during World War I were salvaged during World War II by the blood transfusion units. Thus, many more patients with extensive injuries were operated upon during the Second World War, which may account for the difference in therapy.

Since the Second World War, the results of primary closure in civilian

Table 2. Mortality Rate and Fistula Incidence Following Primary Closure of Colon Injuries in Civilian Practice

		MULTIPLE INJURIES			SIMPLE COLON INJURY		
	YEAR	No. Pts.	Mortality	Fistula	No. Pts.	Mortality	Fistula
Roof[35]	1949–1959	101	10	4	43	0	1
Isaacson[18]	1945–1957	32	1	1	17	0	0
TOTAL		133	11 (8.2%)	5 (3.8%)	60	0	1 (1.7%)
TOTAL (All injuries)		193	11 (5.7%)	6 (3.1%)			

injuries have been excellent. Using the same indications for primary repair as mentioned by Fraser, 193 reported colon injuries have been closed primarily with an overall mortality rate of 5.7 per cent and a fistula incidence of 3.1 per cent (Table 2). In this group, all of the deaths occurred among patients with multiple injuries. It may, therefore, be concluded that primary closure of colon injuries is a relatively safe procedure and should be used unless some contraindication exists.

2. Primary Closure with Proximal Colostomy or Cecostomy

This is indicated for lesions that can be closed primarily but where there is some doubt about the security of the closure. A colostomy diverts the fecal stream from the damaged bowel so that pressure cannot build up within the bowel lumen to blow out the closure. If the repaired area should leak, the colostomy then helps prevent fecal contamination of the peritoneal cavity.

3. Exteriorization

Exteriorization of the colon should be performed when primary closure cannot be done safely. This includes a variety of circumstances and situations:

(a) Extensive damage to the bowel wall
(b) Uncertainty regarding the viability of the bowel wall
(c) When the patient is to be transferred or evacuated to another hospital within a few days
(d) Edematous, inflamed bowel that may not hold sutures well
(e) Exteriorization is most applicable to injuries of the transverse and sigmoid colons. Lesions of the right or left colon are frequently difficult to exteriorize and are better handled by resection.

4. Resection with Colostomy

Resection is indicated when there is extensive damage to portions of the colon that are difficult to exteriorize. Resection was condemned during World War I because it was regarded as too shocking to mobilize so much colon. However, it is fallacious to condemn resection in favor of exteriorization, since mobilization of the same segment of colon is required for either procedure. When extensive colon damage exists, it is no more shocking to resect than to exteriorize, and in some cases, resection is easier. In Ogilvie's series,[26] the mortality rate for colon injuries treated by exteriorization was the same as the mortality rate for those treated by resection with exteriorization of the ends, 64 per cent.

5. Resection with End-to-End Anastomosis

Only a few reported colon injuries have been treated by resection and primary anastomosis. Not enough data are available to evaluate such an

approach. When a resection is necessary, the indications for a primary anastomosis are:

(a) The patient's condition on the operating table is good
(b) The two ends of bowel are unquestionably viable
(c) Anastomosis is possible without mobilizing much additional retroperitoneal bowel
(d) When the cecum has been resected, it is particularly desirable to perform an anastomosis instead of an ileostomy, if at all possible.

Recently, successful experiences have been reported with one-stage resections and primary anastomoses following perforations of the colon. The mortality rate in 56 collected cases was only 3.5 per cent, much lower than that of the standard three-stage resection.[24] This supports the thesis that colon anastomoses may be successfully performed in the unprepared bowel, in the presence of peritonitis. It is possible that, in time, this approach may prove to be the method of choice.

RESULTS

During the past 13 years, all of these techniques have been used at the Colorado General Hospital in managing colon injuries (Table 3).

Table 3. Mortality Rate from 28 Colon Injuries Treated by Various Methods at the University of Colorado Medical Center from 1949 to 1962

METHOD OF REPAIR	NO. OF PATIENTS	NO. OF DEATHS
Primary closure	11	0
Primary closure with colostomy	5	1
Exteriorization	7	0
Resection with colostomy	1	0
Resection with anastomosis	4	2
	28	3 (10.7%)

Two of 19 patients with multiple injuries died of shock; one of nine patients whose only injury was that of the colon died of peritonitis. Although two of the four patients treated by resection with primary anastomosis died, the deaths were due to extensive injury and shock and were not related to the colon injury or its management.

TECHNICAL POINTS

Location of Colostomy

In general, it is preferable to deliver the colostomy through a separate incision. The primary incision is frequently infected in these cases, and

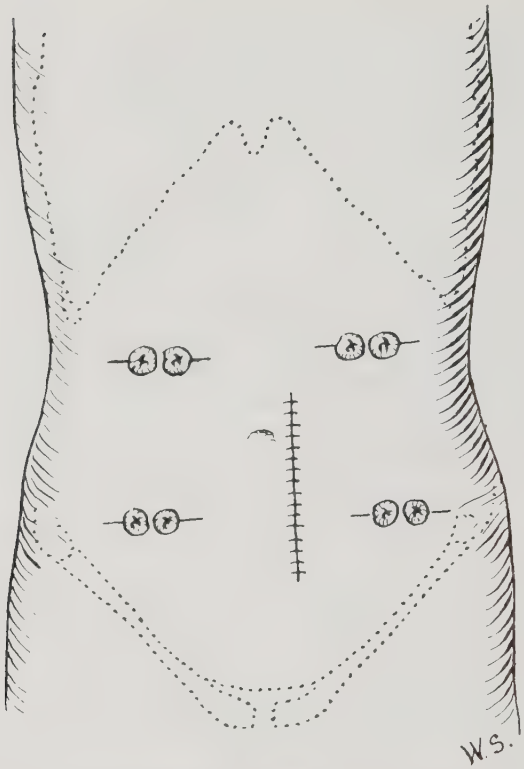

Fig. 1. Preferable colostomy locations.

care of such a wound is greatly handicapped by the presence of a constant source of contamination in its center. The sites for elective colostomies are the centers of the four quadrants so that appliances can fit snugly, away from the umbilicus, the costal margins, and the iliac spines (Fig. 1).

Technique of Colostomy

There are three types of colostomies:

1. *Divided colostomies.* When bowel is resected or when it is easier to divide the bowel to exteriorize the ends, the two ends are brought out through separate stab wounds.

2. *Spur colostomies,* frequently employed in the preantibiotic era, are seldom used today. Their chief advantage is that they can be closed extraperitoneally. In recent years, the risk and complication rate of intraperitoneal colostomy closure has been so low (0.5 per cent) that spur colostomies are rarely needed.[29]

3. *Loop colostomies,* the type used most commonly today, are the easiest to perform. A loop of colon is simply exteriorized and supported on one or two glass rods or a skin bridge. The latter takes a few extra minutes to perform, but has the advantage of permitting complete separation of the loops and total defunctionalization of the distal lumen. The

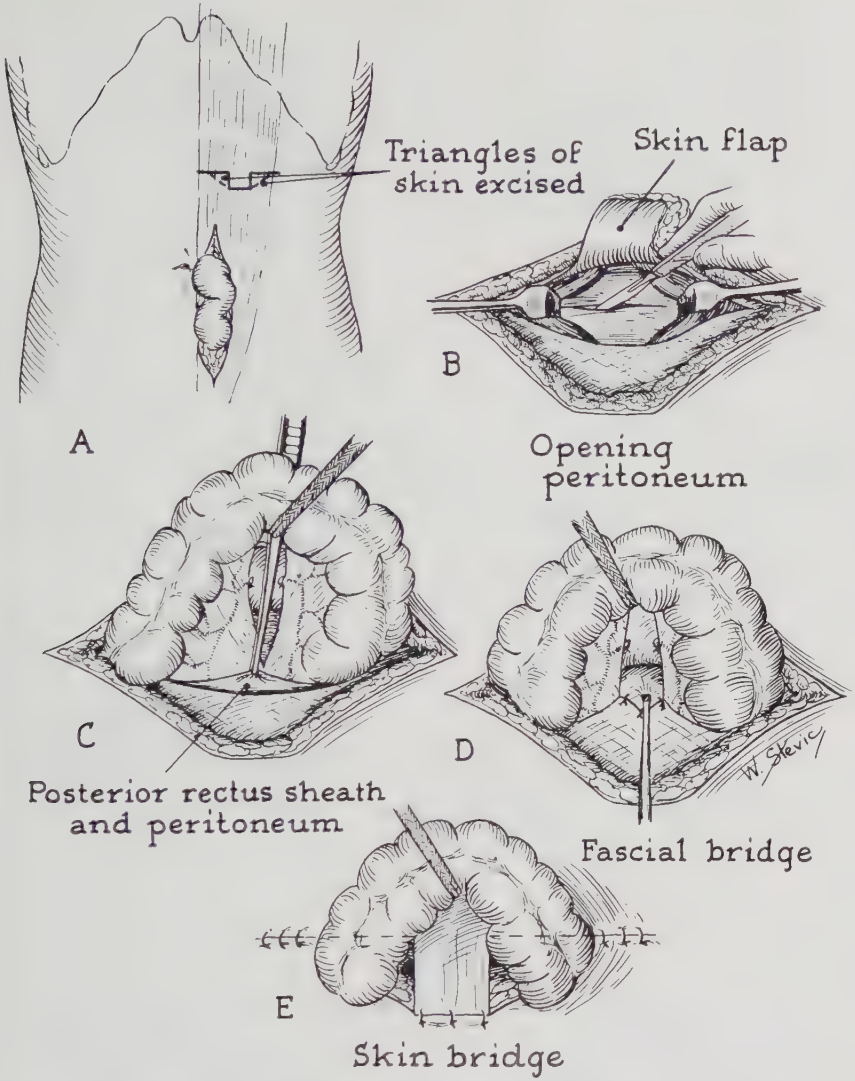

Fig. 2. Technique of loop colostomy. (Adapted from Coller, F. A. and Ransom, H. K.: Carcinoma of the Rectum: Conclusions based on 12 years experience with combined abdominoperineal resection. Surg. Gynec. & Obst. *78:* 304, 1944.)

technique described by Coller and Ransom[7] is illustrated in Figure 2. A hole is made in the mesentery with a vertical opening, dividing the marginal artery (Fig. 2, *C*). This will not compromise the blood supply to the bowel, and it will help avoid serious bleeding later, when the colostomy is divided. One or two terminal arteries to the bowel may be ligated, if necessary. If more are ligated, bowel necrosis may result. The hole in the mesentery should be made large enough to work through,

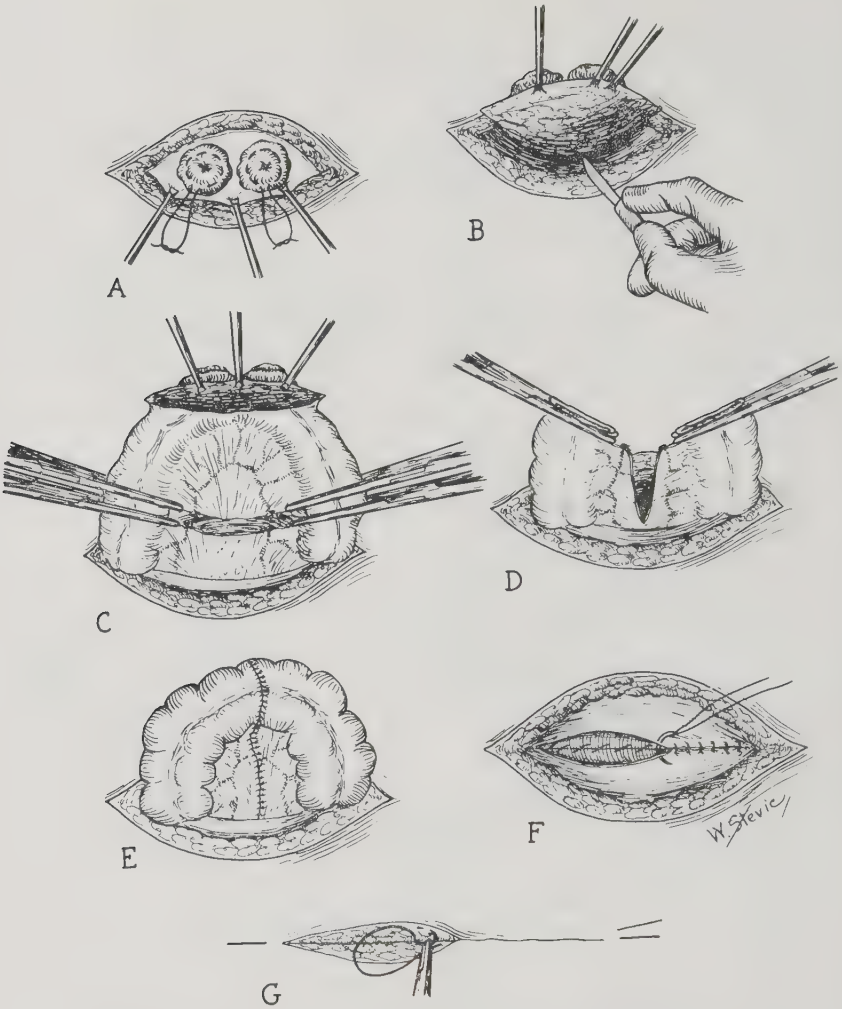

Fig. 3. Technique of colostomy closure.

usually 2 inches or more. The fascial bridge (Fig. 2, *D*) is placed in such a manner as to permit the opening for the proximal colon to admit one finger.

Colostomy Closure

The loop colostomy is best closed intraperitoneally, by formal resection and end-to-end anastomosis. In a follow-up of over 2000 intraperitoneal colostomy closures during the Second World War, Poer[29] reported a

mortality rate of 0.5 per cent. The technique of colostomy closure is shown in Figure 3. To avoid contamination, purse-string sutures are used to close each lumen before the abdomen is prepared. The elliptical skin incision provides margins of skin which can be grasped with Allis or Ochsner forceps to retract the skin over the open bowel mucosa. When the skin flaps are wide enough, it is possible to completely cover the bowel mucosa by approximating the two skin edges with clamps, which are then used as retractors.

The fascial edges are quite adherent to the bowel. They can be separated from it by carefully identifying the bowel and entering the peritoneal cavity at any convenient spot. With a finger inside the peritoneal cavity, the dissection plane between bowel wall and abdominal wall can usually be felt and easily incised. If the bowel is inadvertently opened, it should be closed with a suture. Once freed on all sides, the bowel can be delivered from the abdominal cavity and a formal resection performed. Resection of a wedge of mesentery with the bowel will make closure of the mesentery easier, when the two-layer anastomosis is completed.

Closure of the abdominal wall is not always as clear-cut as shown in Figure 3, *F*. Frequently, the anterior and posterior rectus sheaths and the rectus muscle are scarred together in a single thick layer. Such a wound is best closed with a single fascial layer, using fairly large bites of heavy, nonabsorbable suture material (our preference has been monofilament wire).

Since the incidence of wound infection following colostomy closure is quite high, many surgeons prefer a delayed, primary closure of the skin. The delayed closure, unfortunately, has not solved the problem of wound infections, and does increase the patient's discomfort. For this reason, we have returned to primary closure of most of these wounds.

SUMMARY

Colon injuries should be repaired primarily, if technically possible. Exteriorization or proximal colostomy is indicated when there is extensive bowel damage and the patient's condition is poor. Resection of extensively damaged colon with end-to-end anastomosis may be performed if the patient's condition is good; this is particularly desirable for injuries of the right colon. One technique of loop colostomy and colostomy closure is described.

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The Management of Acute Circulatory Failure

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THE MANAGEMENT of acute circulatory failure is an essential and highly rewarding aspect of trauma therapy. Support of circulatory dynamics is often the only major therapy necessary to restore a critically injured patient to safety. In many other cases the gratifying results of skillfully performed surgery will be realized only if one is successful in maintaining an adequate circulation during the preoperative, operative and postoperative phases of the patient's treatment. Truly then, management of "shock" is a keystone in the overall care of the severely injured.

Definitive care of most traumatic problems may be delayed with safety and advantage to the patient while he is transported to a treatment center where superior facilities and specially trained personnel are available. Shock, however, must be treated as quickly as possible if the injured patient is to have every opportunity for survival. A delay in restoring adequate circulation may prove fatal or lead to serious renal and cardiovascular complications. Therefore, responsibility for resuscitation must be accepted by the first and often the only physician available to the injured patient. For this reason, every physician who may be called upon to care for the victims of trauma, general practitioner and specialist alike, should become competent in handling acute circulatory failure.

INTRODUCTION

The full-blown syndrome of shock is characterized by anxiety, restlessness, pallor, clammy skin, hypotension and thready pulse, and is easily recognized. However, one should be on the alert to identify the more subtle early stages of circulatory inadequacy, indicated by only a mild hypotension, tachycardia or oliguria; even here circulatory failure may be potentially lethal if uncorrected. Prompt recognition and treatment of early circulatory failure will often prevent the development of severe shock with its disastrous complications and high mortality.

Adequacy of circulation is determined from (1) the patient's general appearance; (2) the color, temperature, moisture of the skin; (3) rapidity

of capillary filling of the skin and nail beds: (4) the pulse quality as to fullness, rate and rhythm; (5) the urine volume, preferably hourly; and (6) the arterial pressure relative to the patient's normal range. Primary interest is centered on the effective perfusion of blood through the body tissues, rather than the arterial pressure level. Thus, even if arterial pressure is within a patient's normal range, one should suspect that circulation is marginal or inadequate in the presence of apprehension, narrow pulse pressure, tachycardia, clammy skin with poor color, or oliguria. This is especially true during vasopressor therapy. On the other hand, if a severe vasoconstrictor response, as caused by external cooling, with weak peripheral pulses and cold cyanotic extremities is accompanied by a good urine output, one is reassured that the important central circulation is adequate. Or if the arterial pressure is running lower than normal but the patient has warm, dry, pink skin, with good capillary filling and a good urine output, circulation appears adequate and one need not be unduly alarmed about the relative hypotension. Oliguria and anuria should always make one suspect inadequate circulation. However, if anuria is accompanied by a normal arterial pressure, a strong full pulse, warm dry skin and good venous filling, and the patient appears comfortable and relaxed, the anuria is probably due to renal insufficiency rather than poor tissue perfusion. Thus each of the different bedside observations with which we estimate the adequacy of circulation has limitations, and one should not place absolute confidence in any one of these observations when taken out of context with the overall clinical picture.

Unlike experimental traumatic and hemorrhagic shock in the laboratory, in which hypovolemia usually plays an important intermediate role, circulatory failure in patients following trauma may arise from a variety of causes or combinations of causes, including pre-existing cardiac and pulmonary disease. Therefore, proper treatment will vary markedly from case to case. From a clinical standpoint, we can hardly limit our attention to so-called traumatic shock, but must consider the treatment of circulatory failure in general as it may occur in injured patients.

In most cases, the cause for circulatory failure is easily recognized and specific therapy readily restores the circulation to normal. Straightforward cases such as simple hypovolemia or uncomplicated pneumothorax usually offer no real difficulty in treatment because they are *diagnosed correctly and treated promptly*.

Less frequently, one encounters cases in which initial supportive efforts fail to improve circulatory dynamics and the cause for persistent hypotension remains obscure. These patients are in a precarious state, and survival depends upon accurate and aggressive therapy. Uncertainty in treatment increases the hazard of a fatal outcome, so therapy must be directed with confidence.

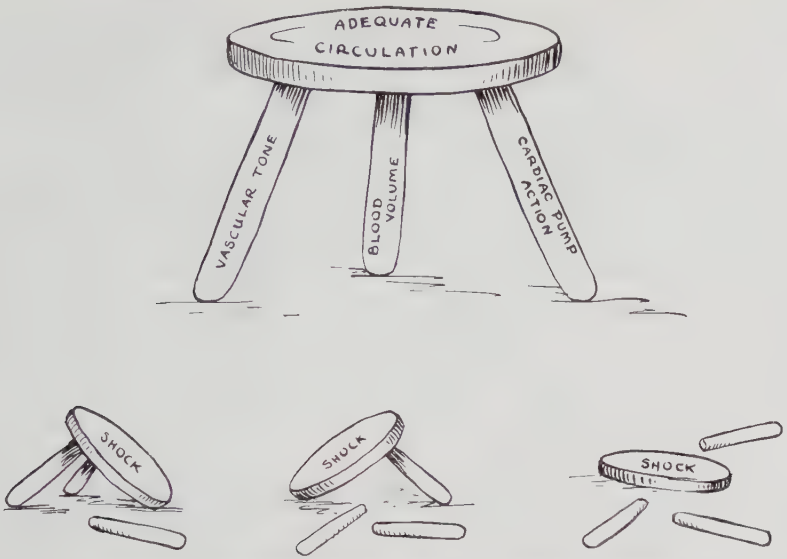

Fig. 1. Adequate circulation depends upon an effective combination of blood volume, cardiac pump action and vascular tone. Shock may result from a deficiency in one or more of these three factors. (Wilson et al., Arch. Surg. *85:* 563, 1962.)

The following discussion is especially concerned with difficult shock problems with the idea that the physician who has a sound approach to complicated circulatory failure will have little trouble in handling the simpler routine cases.

PHYSIOLOGIC CONSIDERATIONS

Effective and decisive management of severe circulatory failure requires an understanding of the functional composition of the circulatory system and the significance of the central venous pressure (CVP).

The Functional Composition of the Circulatory System

It is helpful to think of the essential components of the circulatory system as the props of a three-legged stool (Fig. 1). Stability of the stool—adequate circulation—depends upon the combined support of: (1) cardiac pump action, (2) circulating blood volume and (3) vascular tone. Circulatory failure may result from a deficiency of any one of these factors or, more important, it may result from a combination of deficiencies of any two or all three of these factors occurring simultaneously.

When circulatory failure is due to multiple defects in the circulatory system, therapy becomes correspondingly more difficult. This is best illustrated by the clinical example of a patient in shock from obvious blood loss, with hypoxic depression of myocardial function due to splint-

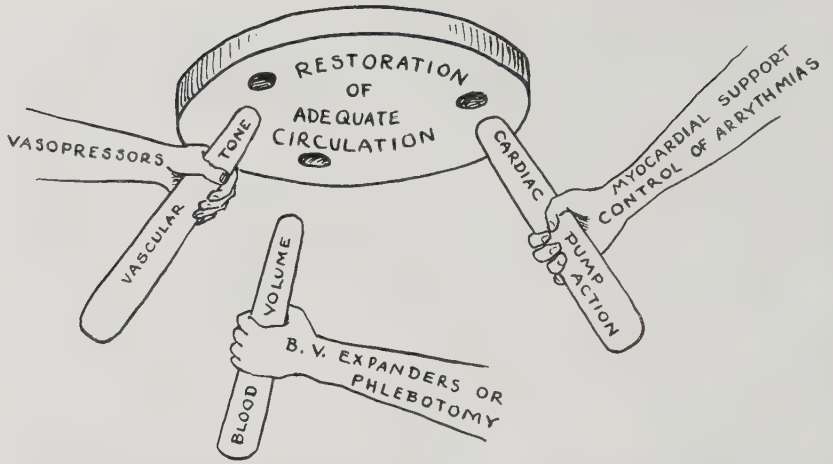

Fig. 2. Restoration or maintenance of adequate circulation requires a coordinated support of blood volume, cardiac pump action and vascular tone. (Wilson et al., Arch. Surg. *85:* 563, 1962.)

ing of painful rib fractures. If the physician recognizes only the hypovolemic cause for shock, he will rightly plan to replace blood volume until arterial pressure is restored to normal. Consequently, after adequate blood volume has been replaced, persistent hypotension from defective cardiac pump action may lead the physician to continue blood volume replacement, overload the failing heart, and risk fatal pulmonary edema. If, on the other hand, the physician were treating the same patient with the added safety afforded by central venous pressure monitoring, he could recognize when hypovolemia was no longer the cause of the persistent hypotension by a rising and elevated CVP. Then, even though the arterial pressure was low, he could stop blood volume replacement with confidence and direct his attention to improving cardiac pump action, in this case by improving ventilation. Thus the problem with complicated shock is not which single factor to treat, but rather how to coordinate the support of multiple factors simultaneously (Fig. 2).

The analogy of the three-legged stool illustrates the essential role of blood volume, cardiac pump action and vascular tone in the circulatory system as a whole. However, unlike the rigid wooden legs, each of these essential components may undergo considerable alteration and still support an adequate circulation as long as there are satisfactory compensatory alterations in the other components. The circulatory system is extremely flexible and may accomplish its purpose—adequate perfusion of blood through the body tissues—by any *effective combination* of (1) the central pump action of the heart, (2) the volume of blood propelled by the pump, and (3) the degree of vasoconstriction or vasodilatation present. The latter determines (*a*) the volume of the vascular

system in which the blood is contained, and (*b*) the resistance to flow of blood through the vascular system.

Shock or any lesser degree of circulatory failure represents a failure of the circulatory system to establish an effective combination of cardiac action, blood volume, and vascular tone. Restoration and maintenance of an adequate circulation requires a consideration of all three of these components, first because more than one component may be defective, and second because the optimum of any one of these components will depend upon the limitations or alterations of function of the other two components. The second point is particularly true in maintaining an optimal blood volume. Effectiveness of the blood volume depends upon the pumping action of the heart and the vascular tone, and adequacy of the blood volume depends upon the volume of the vascular system which must be filled. Therefore a physiologically optimal blood volume is defined by the functional capacity of the combined cardiac and vascular organs rather than by a fixed percentage of the patient's body weight or even his usual blood volume during normal circulatory dynamics. Also one may expect the optimal blood volume to be redefined by alterations of cardiac and vascular function during the transition from circulatory failure to normal circulatory dynamics. In the patient described above, the optimal blood volume during shock from blood loss and hypoxic cardiac failure might be hypovolemic relative to his blood volume in good health, and immediate restoration of this normal blood volume might be ineffective and hazardous. However, as ventilation and oxygenation improve myocardial function, the optimal blood volume will increase, and the patient may then require and safely tolerate further blood volume replacement.

Significance of the Central Venous Pressure

The venous pressure originates from the arterial pressure transmitted through the capillary bed into the venous reservoir, and the central venous pressure (CVP) is then determined by the interrelated effects of *the same three factors which comprise the circulation*—cardiac pump action, circulating blood volume and vascular tone.[3, 4, 5]

If cardiovascular dynamics remain constant, the CVP will vary directly with the circulating blood volume. If blood volume and vascular tone remain constant, the CVP will vary inversely with the cardiac pump action. To a lesser extent, the CVP will vary with alterations in vascular tone, decreasing with vasodilation. The CVP is not an index of blood volume per se, nor of cardiac output per se, but if one appreciates the interrelated effects of blood volume, cardiac action and vascular tone, one may think of the CVP as an index of the circulating blood volume relative to the cardiac pump capacity, or, more accurately, relative to the

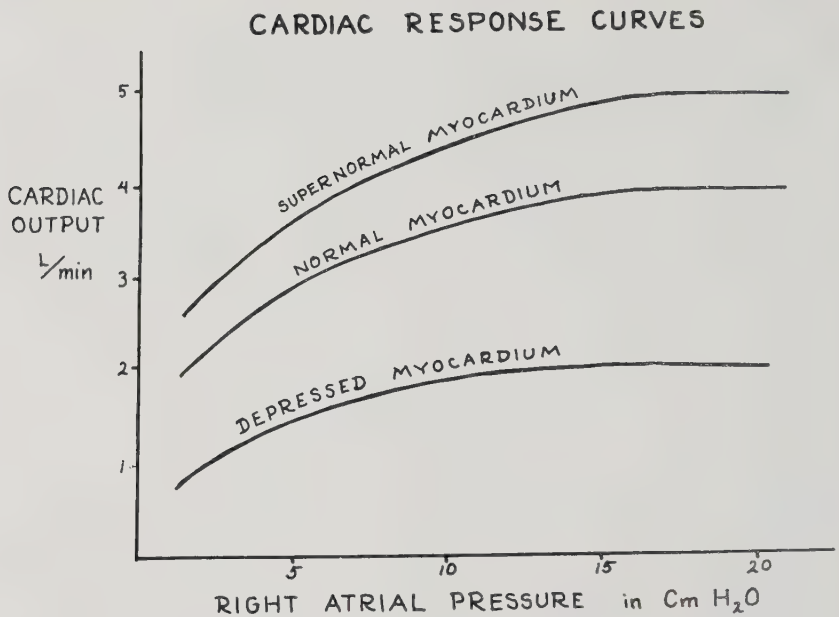

Fig. 3. The work of the ventricle for any given filling pressure varies with the condition of the myocardium.

total cardiovascular capacity. Thus, if one is confronted with hypotension and a CVP which is not elevated, the circulating blood volume, regardless of the measured quantity, is low relative to the cardiac pump capacity, and blood volume expanders can be given safely and effectively to improve cardiac output. If, on the other hand, one is confronted with hypotension and a CVP that is elevated and rising, *and mechanical obstruction to venous return is ruled out,* then the circulating blood volume present, regardless of the measured quantity, is high relative to the cardiac pump capacity, and further administration of blood volume expanders becomes progressively less effective in improving cardiac output, and carries an increasing risk of overloading a failing myocardium or producing pulmonary edema.

These concepts are further illustrated by a consideration of Starling's Law of the Heart, in which the energy of ventricular contraction, however measured, is a function of the length of the ventricular muscle fiber before contraction.[8]

In Figure 3, the relationship between ventricular work and the diastolic length of the ventricular muscle fibers is reproduced as several ventricular function curves, or cardiac response curves, each obtained under different circumstances. In these function curves, the ventricular work is expressed as cardiac output, and the diastolic length of the muscle fibers is expressed as the filling pressure of the heart, or the central venous pressure. Inspec-

tion of the ventricular function curve of the normal myocardium reveals that when the filling pressure of the heart is low there is a large reserve in the cardiac pump capacity and that the strength of myocardial contraction, and therefore cardiac output, can be improved by increasing the CVP with an increased venous return, thereby increasing the diastolic length of the ventricular muscle fibers. As the CVP increases, the strength of the myocardial contraction and cardiac output increase to a point where further increase in CVP no longer increases the myocardial work and cardiac output.

Sarnoff has emphasized a most important refinement of this Frank-Starling relationship by pointing out that a myocardium does not operate on only one ventricular function curve, but actually operates within a family of ventricular function curves,[7] as illustrated in Figure 3. Thus, stimulation of the myocardium may increase contractility and shift myocardial performance to a more efficient supernormal ventricular function curve in which there is a correspondingly stronger myocardial contraction for any given filling pressure. Depression of the myocardium from hypoxia, fatigue or biochemical derangement decreases contractility and shifts myocardial performance to a less efficient ventricular function curve in which the strength of myocardial contraction is considerably less for any given filling pressure than when the myocardium is operating on its normal ventricular function curve. Therefore, if the CVP is elevated in the presence of circulatory failure and there is an adequate heart rate with no mechanical obstruction to venous return, one has by definition a failing myocardium performing on a depressed ventricular function curve. Here no amount of additional blood volume will improve the cardiac output, and only by improving performance of the myocardium itself (i.e., shifting to a more efficient ventricular function curve) can one expect to improve the circulation.

The elevated CVP thus provides a physiologic indication to withhold blood volume expanders and directs the physician's attention to improving cardiac pump action. Only after myocardial function is improved and cardiac pump capacity is thereby increased, as reflected by a lowering of the CVP, could further blood volume expanders be expected to increase cardiac output.

MECHANICS AND INTERPRETATION OF THE CENTRAL VENOUS PRESSURE

The peripheral venous pressure is not a reliable index of central venous pressure (CVP). In experimental circulatory overloading[11] and in congestive failure,[6, 11] the normal gradient between peripheral and central venous pressure diminishes and approaches zero. In a significant number of both experimental and clinical studies, this gradient has even reversed

so that definitely elevated CVPs were associated with normal peripheral venous pressures. Therefore, the only venous pressure determination which is an accurate index of circulatory dynamics is one in which the tip of the indwelling venous catheter is in direct fluid continuity with the right atrium without intervening venospasm or venous valves which might interfere with the direct transmission of right atrial pressure to the catheter tip. This CVP may be measured anywhere in the superior or inferior vena cava or their immediate valveless tributaries, i.e., the innominate or common iliac veins. Cannulation of the superior vena cava system through a percutaneous puncture of the subclavian or internal jugular veins carries the least risk of thrombo-embolic complications and avoids false high readings which may occur in the inferior vena cava with abdominal distention.

A detailed account of the technique and application of CVP monitoring has recently been published.[12] In essence, the manometer is constructed by connecting an empty intravenous administration tubing as a side arm into the regular intravenous administration system with a three-way stopcock or with a No. 18 needle in piggyback fashion.

The manometer tube and a centimeter scale are then taped to a vertical intravenous stand, with the zero point positioned at the mid-axillary line in the supine position. The CVP is determined by connecting the manometer tube in continuity with the indwelling venous catheter and temporarily stopping the intravenous infusion; this is accomplished by a simple turn of the three-way stopcock or, in the case of the piggyback arrangement, by clamping the intravenous administration tubing between the infusion bottle and the point where the two tubings are connected. All determinations must of course be made with the patient in the same position relative to the manometer scale.

The CVP is an important adjunct in the evaluation and management of the patient in shock. However, the goal is not to treat the venous pressure alone any more than one would treat the arterial pressure or the urine output without consideration of the overall clinical picture. One should not try to maintain any particular venous pressure level, and as long as there is adequate circulation the CVP level is relatively unimportant. However, when circulation fails, the CVP becomes extremely helpful in planning how to improve circulatory dynamics. In this respect, the trend of the CVP and especially the response to therapy are often more important than any isolated CVP reading.

AN OUTLINE OF MANAGEMENT FOR THE PATIENT IN SEVERE CIRCULATORY FAILURE

The key to resuscitation of the severely injured patient is an accurate diagnosis of the cause for the circulatory failure. Therefore, therapy

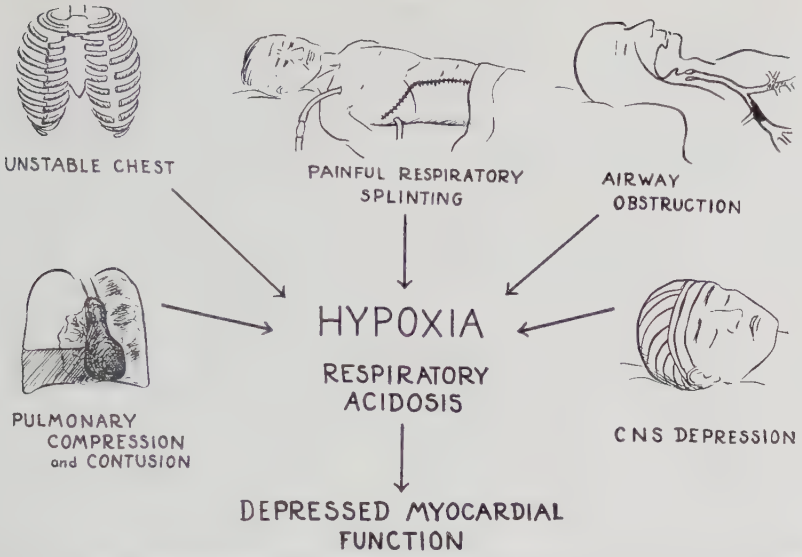

Fig. 4. Many complications of trauma lead to hypoxia and respiratory acidosis, which depress myocardial function.

starts with a rapid but thorough search for signs of inadequate respiration, impaired venous return, blood loss, heart failure, and sepsis. In the early hours following injury, acute hypovolemia and respiratory insufficiency will account for the great majority of shock problems. Mechanical obstruction to venous return and primary cardiac failure are important but less common causes of shock in the immediate post-injury period.

In the days and weeks to follow, infection and cumulative losses of body fluids and electrolytes may develop, and sepsis and biochemical derangements will play an increasing role in the genesis of circulatory failure.

The following steps in management of severe circulatory failure are a guide to evaluation as well as therapy.

1. Adequate Respiratory Function

The importance of deficient ventilation and oxygenation cannot be too strongly emphasized as an etiologic factor of shock. It is much more common than generally appreciated and may be overlooked when it accompanies other more obvious problems. Deficient respiratory function may result from a variety of conditions related to trauma. The common causes are presented in Figure 4. They include (*a*) splinting of ventilatory motion because of pain from abdominal or thoracic incisions, rib fractures, pleuritis or peritonitis, (*b*) airway obstruction from retained bronchial secretions and aspirated blood or gastric contents, (*c*) unstable

Fig. 5. Inadequate ventilation and inadequate circulation, separately or in combination, can lead to progressive circulatory failure.

chest from multiple fractures of the thoracic cage, (*d*) pulmonary compression from pneumothorax and/or hemothorax, (*e*) abdominal distention, (*f*) pulmonary contusion, and (*g*) hypoventilation secondary to central nervous system depression from brain trauma, medications, or anesthetic agents. All of these conditions can produce hypoxia and respiratory acidosis from retention of CO_2, which in turn may depress myocardial function. If the subsequent reduction in cardiac output is significant, it will lead to a metabolic acidosis which will further depress myocardial function and thus perpetuate a vicious circle leading to severe shock and death (Fig. 5). The hazard of these respiratory complications is of course increased when they occur in combination and in patients with underlying pulmonary disease.

Clinical evaluation of the severely injured begins with a careful check for airway obstruction, depressed breath sounds, tracheal deviation, and cyanosis. Correction of hypoxia or respiratory acidosis may require immediate tracheal intubation, endobronchial suctioning, insertion of chest tubes, ventilatory support with a mechanical respirator, or oxygen therapy before blood volume requirements can be evaluated accurately. Pulmonary infection may require antibiotics, and bronchial spasm or edema may require bronchodilator and steroid therapy. A myocardium depressed from hypoxia may be laboring at its peak capacity even though the actual blood volume is low, and only after myocardial function is restored to normal by improved ventilation and oxygenation will the cardiac pump capacity be adequate to handle the needed blood volume replacement.

Ventilatory difficulties which are handled satisfactorily by the patient initially may become a serious problem as the patient weakens from fatigue. When increasing tachycardia and cyanosis are associated with inadequate ventilation or more labored ventilatory efforts, mechanical respiratory support should be considered. Early use of either intermittent or continuous positive pressure ventilation in such cases will usually result in improved skin color, a slower stronger pulse, clearing of expiratory wheezing, and a restful sense of well-being. Thus appropriate support of respiratory function may prevent deterioration of biochemical homeostasis and progressive circulatory failure.

In cases of severe respiratory insufficiency, ventilation can be effectively supported with a positive pressure respirator connected to a tracheostomy or endotracheal tube fitted with an inflatable rubber cuff. Positive pressure ventilation and bronchodilator medication will usually overcome bronchospasm and expiratory wheezing. Occasionally apprehension and struggling for air interfere with efficient support from the respirator. Such deleterious fighting against the respirator can sometimes be controlled only with fairly deep narcosis or sedation and hyperventilation, which will permit the respirator to take over respiration completely. The usual danger of oversedating these pulmonary cripples with respiratory depressant drugs does not apply to a patient carefully controlled on a mechanical respirator.

The dramatic improvement in ventilation, color and pulse quality, and the relief of frightening air hunger following aggressive and effective respiratory support are among the most impressive of recent advances in resuscitation.

2. Mechanical Obstruction to Venous Return

Obstruction to venous return in the mediastinum is a less common cause of circulatory failure, but should be kept in mind to assure recognition of this serious complication when it does occur. Physical examination should include a check for cardiac displacement, tracheal deviation, increased venous pressure, and pulsus paradoxicus.

In view of our interest in the CVP, it is particularly important to rule out obstruction to venous return as a cause for an elevated CVP before assuming that circulatory failure is due to intrinsic failure of the myocardium. Interference with cardiac filling from pericardial tamponade or mediastinal compression must be corrected by aspiration or thoracotomy before the cardiac pump can utilize the blood volume already present in the venous reservoir for effective cardiac output.

3. Maintenance of Optimal Blood Volume

The foremost question when confronted with circulatory failure is whether to administer blood volume expanders or support cardiovascular

function with drugs, that is, whether the problem concerns hypovolemic or nonhypovolemic circulatory dynamics. Most commonly an injured patient with hypotension is suffering from hypovolemia. In gauging blood volume replacement, a moderate blood pressure depression may represent a blood volume depletion as great as 35 per cent, and severe shock with absent pulses may represent a blood volume depletion of 50 per cent or more. Occasionally the compensatory vasoconstriction accompanying blood loss is so effective that arterial pressure and pulse are stable until precipitous and dangerous circulatory decompensation takes place. Obviously, blood volume replacement cannot be guided by visible losses alone, for external losses before hospital admission and internal losses from bone fractures and ruptured viscera may be very large.

In general, if hypotension is due to hypovolemia alone, arterial pressure will respond quickly to rapid blood volume replacement even if there is still a significant residual blood volume deficit. The vasoconstrictor response to hypovolemia converts the vascular tree to a more rigid hydraulic system, in which a relatively small increase in fluid volume is able to effect a considerable increase in pressure. Therefore, if arterial pressure fails to show any improvement after the rapid administration of 500 to 750 cc. of blood volume expander in an adult (10 per cent of the patient's estimated blood volume) one should suspect that the persistent hypotension is probably due to a cause other than pure hypovolemia.

It is often impossible to differentiate hypovolemic from nonhypovolemic hypotension by physical examination alone. Acute cardiac failure may present with apprehension, pale clammy skin, clear lung fields on auscultation, collapsed peripheral veins, and absent or thready pulse, thus simulating perfectly the appearance of hypovolemic shock. Moderate hypovolemic hypotension may present with warm pink skin and good peripheral venous filling—apparently because of reflex vascular dilation. The clinical differentiation between hypovolemic and nonhypovolemic circulatory failure is further complicated when cardiac disease and blood loss occur together. Unrecognized internal hemorrhage may be the underlying cause of shock in a patient with known longstanding heart failure or with an obvious fresh myocardial injury. Likewise unrecognized cardiac failure may be the underlying cause of shock in a patient with obvious active bleeding. Finally, chronic lung disease, pneumonia or tracheobronchial contents from aspiration or retained secretions can produce cyanosis, wheezing, rhonchi, auscultatory rales and tachycardia indistinguishable from cardiac failure. Under such perplexing circumstances, the clinician must maintain a blood volume adequate to prevent the complications of hypovolemia and at the same time not overload a failing heart pump with excessive blood volume and

thereby increase the risk of pulmonary edema. A practical and dependable method of differentiating between hypovolemic and nonhypovolemic circulatory failure is necessary to accomplish this difficult and essential objective.

Central venous pressure monitoring is a simple and readily available procedure which permits making the critical distinction between hypovolemic and hypervolemic circulatory dynamics at the bedside. When associated with circulatory failure or shock, an unelevated CVP (0 to 5 cm.) indicates hypovolemic circulatory dynamics (not necessarily measurable hypovolemia) in which the circulating blood volume present is well within the cardiac pump capacity. Therefore blood volume expanders may be given safely and with expectation of improving the circulation regardless of the measured blood volume. If the CVP remains unelevated during massive blood volume replacement, the heart is not being overloaded and aggressive blood volume replacement may be continued with confidence, thus preventing the serious cardiovascular-renal complications associated with prolonged hypovolemic hypotension. When hypotension continues or recurs despite large blood volume replacement, a persistent normal CVP suggests continuing blood volume depletion, and surgical exploration should be considered for control of obscure bleeding.

Inadequate circulation with intermediate CVP levels (8 to 12 cm.) may require a trial of blood volume expanders with careful observation of the urine output and the arterial and central venous pressure response to therapy.

On the other hand, when obstruction to venous return is ruled out, a significantly elevated and rising CVP (15 to 20 cm.) indicates hypervolemic circulatory dynamics (not necessarily measurable hypervolemia). In circulatory failure with hypervolemic dynamics, the circulating blood volume present is already encroaching on the reserve of the heart pump and, regardless of the measured blood volume, further blood volume replacement is unlikely to improve the circulation and may be hazardous. The elevated CVP thus provides a physiologic indication to withhold further blood volume expanders and improve circulation by improving the cardiac pump action. After the heart action has improved, the CVP may fall toward normal, indicating that the cardiac pump capacity has increased and that further blood volume expanders may now be given safely and effectively. In such a manner, continuous observations of the CVP may serve to integrate optimal blood volume maintenance with pharmacological support of cardiovascular function.

4. Support of the Cardiac Pump

If circulatory failure is associated with hypervolemic dynamics, attention should be directed to improving heart action with due con-

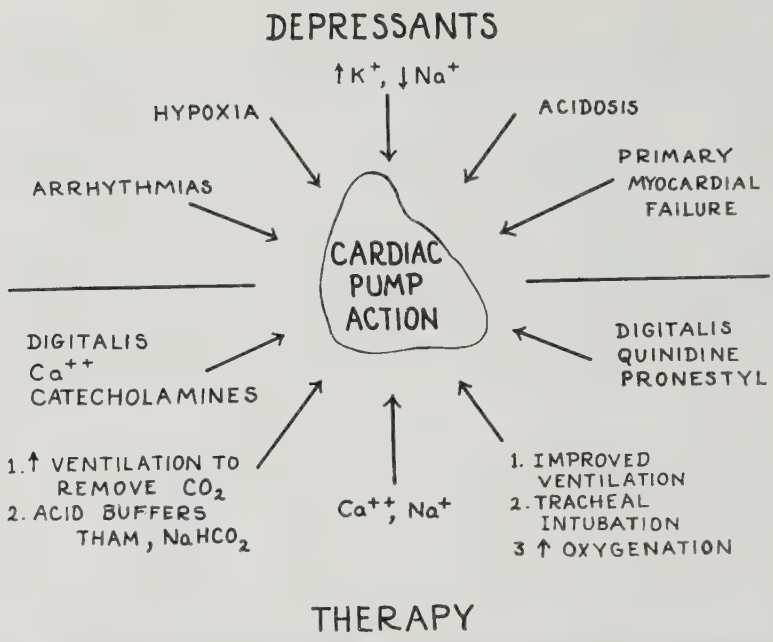

Fig. 6. All of the many cardiac depressants must be considered and corrected for effective support of cardiac pump action.

sideration of all the possible causes for deficient cardiac function. In Figure 6, the common causes of inadequate cardiac pump action are grouped above as depressants, and the corresponding therapeutic measures to correct these depressant actions are listed below.

(*a*) The important and very common occurrence of *hypoxia* has been discussed above under Maintenance of Adequate Respiratory Function. An important but seldom appreciated benefit of respiratory support is the actual decreased work load on the heart achieved by taking over the work expended on ventilation by the dyspneic patient. There are certain theoretical objections to using positive pressure respiratory support because of the interference with venous return. However, one must weigh this inconvenience to the heart pump against the improvement in cardiac action which may result from the increased oxygenation of the myocardium. In general, the CVP will rise only a few centimeters with positive pressure ventilation, and when increased oxygenation causes a marked improvement of circulation the CVP may fall as much as 10 cm. as the arterial pressure and urine output increase, indicating the net gain in cardiac efficiency despite the positive pressure respirator.

(*b*) *Arrhythmias* may reduce the cardiac pump efficiency independently of myocardial function. Recognition of the arrhythmia, evaluation with an electrocardiogram, and proper therapy with digitalis, quinidine,

pronestyl or occasionally Isupryl, should control arrhythmias without difficulty in most cases.

(*c*) *Electrolyte derangements* may also decrease myocardial function. Hyperkalemia is particularly prone to occur during multiple blood or plasma infusions, and for this reason the prophylactic administration of intravenous calcium during such circumstances seems warranted. Reduction in urine output will of course increase the hazard of a build-up of potassium in the serum. Concomitant hyponatremia can potentiate the effects of elevated serum potassium or may itself be a depressant. Electrocardiographic tracings and serum electrolyte determinations are occasionally necessary to guide clinical therapy. The use of calcium, glucose and insulin, sodium, and carboxylic acid sodium exchange resin have practically eliminated the need for artificial dialysis to reduce an elevated serum potassium in acute situations.

(*d*) *Acidosis*, either respiratory or metabolic in origin, can depress myocardial function. The etiology of respiratory acidosis has been discussed in the section on respiratory support. Metabolic acidosis may develop fairly rapidly from the accumulation of acid metabolites produced by the tissue hypoxia accompanying inadequate circulation. The inadequate circulation may be secondary to myocardial depression from respiratory acidosis, or it may be primary as a result of hypovolemia or cardiac failure. As depicted in Figure 5, either inadequate respiration alone or inadequate circulation alone can lead to progressive circulatory failure through the cycle of depressed myocardial function which leads to inadequate circulation which increases the metabolic acidosis which further depresses myocardial function.

Arterial pH, pCO_2 and CO_2 content determinations are indicated whenever circulatory failure persists after establishing adequate ventilation and an optimal blood volume. It is often surprising how serious the pH depression may be while the patient's clinical appearance is deceptively stable, especially while he is on vasopressor therapy. However, rapid deterioration may occur at any moment if vigorous corrective measures are not taken. For those who do not have access to arterial pH, CO_2 and CO_2 content determinations, it should be remembered that a normal CO_2 content alone is no assurance that serious acidosis is not present. This is because the combination of a respiratory acidosis from retention of CO_2 and a metabolic acidosis which decreases the CO_2 may present with a normal CO_2 even though there is a marked depression of the pH. Therefore measurements of CO_2 are reliable as an index of metabolic acidosis only if respiratory function is perfectly adequate. In complicated cases of shock with concomitant pulmonary insufficiency, pH measurement with pCO_2 and CO_2 content determinations are necessary to identify accurately the serious biochemical derangements that may need correction.

The correction of respiratory acidosis requires improvement of ventilation so as to increase the excretion of CO_2 from the lungs. When pulmonary insufficiency limits ventilatory excretion of CO_2, the acid buffer THAM (2-amino-2-hydroxymethyl-1,3-propanediol) is helpful in raising the pH and lowering the elevated pCO_2. The associated increase in bicarbonate is available for excretion by the kidneys. Correction of respiratory acidosis may itself improve cardiac action with subsequent improvement of concomitant metabolic acidosis. Spontaneous correction of mild metabolic acidosis may follow simple restoration and maintenance of adequate circulation by proper blood volume replacement and cardiac support. Severe metabolic acidosis should be treated with sodium bicarbonate to insure more adequate myocardial function.

Those who do not have pH and pCO_2 determinations available will do well to remember the importance of acid-base imbalances and how to prevent them. Appreciation of the crucial role of respiratory function and vigorous respiratory support will often be life-saving, and the judicious use of sodium bicarbonate or THAM in shock states on an empiric basis may be advisable. Determinations of pH and pCO_2 should be repeated at intervals, if possible, to check the progress of therapy until the pH is restored to safe levels and ventilatory function is proved adequate by a normal pCO_2.

(*e*) When high venous pressure shock persists despite adequate oxygenation, an effective rhythm, normal acid-base balance and normal electrolytes, one must conclude that there is *primary failure of the myocardium.* Digitalization is indicated, and is particularly effective in the presence of tachycardia when it can restore a more efficient cardiac rate as well as improve myocardial contractility. Intravenous calcium has a potent effect on increasing myocardial strength and has the advantage of immediate effect. However, one must keep in mind its tendency to increase myocardial irritability and to potentiate the effect of digitalis. Thus calcium should be used carefully, with a constant watch for arrhythmias, especially during concomitant digitalization. A third type of drug which can improve myocardial contractility is epinephrine and related agents.[7, 9] Figure 3 shows the middle and lower cardiac response curves of the normal and the diseased myocardium. The upper cardiac response curve demonstrates the "supernormal" myocardial response to epinephrine and other inotropic catecholamines. In the supernormal state there is greater myocardial contractility (reflected by increased cardiac output) for any given diastolic length of the myocardial fibers (reflected by filling pressure, CVP). In clinical practice one is not concerned with obtaining a supernormal cardiac performance, but when the myocardium is clearly performing on a diseased myocardial function curve, as evidenced by circulatory failure with a high CVP, one should

consider utilization of the myocardium-stimulating properties of sympathomimetic agents to shift myocardial performance to a more normal ventricular function curve. Consideration of catecholamines as myocardial stimulants rather than vasoconstrictor agents is an important point and leads to the next subject, that of vasopressor therapy.

VASOPRESSOR THERAPY AND VASCULAR TONE

Considerable disagreement continues as to the merits and hazards of vasopressor therapy. Despite excellent animal studies that demonstrate the deleterious effects of pressor amines, there is a strong clinical impression that vasopressor agents have been life-saving in some cases of severe circulatory failure. In any event, safe and effective use of vasopressors depends upon a rational selection of patients and a clear knowledge of the pharmacologic action of the agent used.

Pressor amines may act both peripherally on the vascular bed and centrally on the heart itself. Each agent must be evaluated with regard to its capacity to produce vasoconstriction, increase heart rate and improve the contractility of the myocardium.

Vasodilatation associated with normal blood volume and cardiac action causes a drop in CVP with a reduction in venous return.[11] Under such circumstances a pure vasoconstrictor may improve the circulation by restoring a proper relationship between the volume of the vascular bed and the blood volume present. However, such a disproportion between blood volume and vascular bed volume with a low CVP can also be improved by blood volume expanders, for the defect is in fact a low circulating blood volume relative to the total cardiovascular capacity.[11]

On the other hand, pure vasoconstrictor action would appear undesirable if the CVP is elevated. First, the increased peripheral resistance from arterial constriction will increase the work load on the already failing myocardium and actually interfere with rather than aid the flow of blood through the vascular bed. Second, the increased venous return from constriction of the venous reservoir can hardly help cardiac output, since the venous return is already high relative to the cardiac pump capacity. And yet many patients in severe circulatory failure with apprehension, pale and clammy skin, marked oliguria and an elevated CVP have responded dramatically to certain catecholamine agents such as epinephrine, norepinephrine or Aramine (metaraminol) when all other forms of therapy failed. Improvement of circulation in these patients was unquestionable in that the increase in arterial pressure was associated with a sense of well-being, warm, dry, pink skin, increase in urine output, and a decrease in the CVP. It appears therefore that in such cases vasopressor therapy has improved cardiac output not by vasoconstrictor

effect but by shifting from an inefficient ventricular function curve, in which cardiac output was inadequate despite an elevated CVP, to a more favorable one in which cardiac output is adequate with an equal or even lower CVP. This interpretation is further supported by experience with some cases of severe circulatory failure which failed to respond or deteriorated with Neo-Synephrine, a pure vasoconstrictor, and yet responded well to noradrenalin or Aramine, which are known to have inotropic effects.

With these thoughts in mind, one might approach the use of vasopressors along the following lines. When circulatory dynamics do not respond satisfactorily or remain stable after initial therapy with the milder vasopressor agents, CVP monitoring should be instituted before embarking on a prolonged course of vasopressor support, especially with the more potent pressor agents. If the CVP is low, blood volume expanders should be tried in preference to vasopressors in order to avoid the administration of vasopressors during unrecognized hypovolemia—the chief hazard of vasopressor therapy. A large percentage of patients placed on vasopressor therapy for suspected normovolemic circulatory failure will prove to have a low CVP, and subsequent administration of blood volume expanders frequently permits complete withdrawal of the vasopressor agent.

If arterial hypotension persists with a rising elevated CVP, pressor amines with inotropic action may be considered along with other means of supporting myocardial function. These agents should be used in as small quantities as possible, maintaining arterial pressure only at lower normal levels for a given individual. Attempts should be made to withdraw or reduce the pressor agent as soon as possible with continuous monitoring of the CVP, which may fall during vasopressor therapy. A drop in CVP indicates that cardiac pump capacity has increased relative to the available blood volume, so that further blood volume expanders may be given safely and may decrease the requirement for the vasopressor agent.

When vasopressors must be used, consideration should be given to the possibility of sepsis. Antibiotic coverage must be adequate. Correction of acidosis and the administration of corticosteroids may potentiate the action of catecholamines on vascular tone.

Summarizing these basic aspects of myocardial support, it should be emphasized that an elevated CVP is not a specific indication for any particular supportive measure, but rather a general indication for cardiac support. Due consideration must be given to all the causes of myocardial depression. Stimulation of the myocardium with digitalis or catecholamines will be effective only if oxygenation, acid-base balance and electrolyte concentrations of the myocardium are adequate, and treatment of acidosis will be ineffective if hypoxia remains uncorrected.

CHOICE OF BLOOD VOLUME EXPANDER

The earlier philosophy that "the best replacement for blood loss is whole blood" should be revised in light of our present knowledge. Experimental and clinical studies by Dr. Ben Wilson and others have emphasized the added safety and superior qualities of balanced electrolyte solutions in blood volume replacement as opposed to whole blood transfusions.[2, 10] The advantages of reduced viscosity and prevention of aggregation and sludging of erythrocytes with attendant renal and pulmonary complications have recently been applied to the hemodilution techniques in cardiopulmonary bypass in which 5 per cent glucose, low molecular weight dextran, or serum albumin is substituted for whole blood as a priming agent in the extracorporeal system.

It is well to consider then the 70-kilogram man with a blood volume of 5600 and a hematocrit of 45 per cent who has lost 1000 ml. of blood. If his blood loss were replaced with an isotonic albumin-electrolyte solution, he would have a normal blood volume with a hematocrit of 37 per cent. If a 1500 ml. blood loss were similarly replaced with an isotonic albumin-electrolyte solution, he would have a normal blood volume with a hematocrit of 31 per cent. Even a 50 per cent hemorrhage of 2800 ml. replaced with isotonic albumin-electrolyte solution would result in a normal blood volume and functionally adequate hematocrit of 22.5 per cent. One can tolerate a low hematocrit as long as adequate blood volume and adequate respiratory support are maintained.[13] With these points in mind, there is rarely need for panic in a search for whole blood. The availability of electrolyte solutions and stored serum albumin should make early resuscitation of the injured more convenient and also considerably safer. The ideal agent for emergency blood volume replacement can be obtained by adding 25 grams (two 50-ml. vials) of concentrated serum albumin to each 400 ml. of either isotonic saline or, even better, lactate Ringer's solution. Certainly early blood volume replacement can be initiated with albumin and electrolyte solutions while properly matched blood is being obtained. Blood transfusions should be withheld until indicated by the need for blood volume expansion associated with a significant hematocrit reduction.

THE SIGNIFICANCE OF THE URINE OUTPUT

The urine output is especially helpful as a reflection of the central circulation through the vital organs. During unstable periods, the hourly urine output should be recorded with the other vital signs for closer observation of the patient's condition than is permitted by the usual eight-hour intake-output charting.

Often a decreased urine volume is the earliest sign of hypovolemia

while arterial pressure and pulse are stable. A good urine output is assurance that there is adequate circulation even if the arterial pressure is lower than normal. On the contrary, a poor urine volume does not necessarily mean that circulation is inadequate. Oliguria or anuria usually indicates inadequate arterial pressure or inadequate blood volume, but derangements of blood chemistries and true renal insufficiency must also be considered. The significant point is not the reduced urine volume itself, but the possibility that the oliguria is due to inadequacy of the general circulation. There is no immediate risk associated with renal insufficiency as long as respiratory and circulatory function are intact. However, if oliguria or anuria is due to poor renal perfusion from general circulatory inadequacy, the patient is threatened with progressive deterioration of circulatory function unless corrective measures are taken promptly. Too often oliguria is attributed to kidney failure and believed to be the cause of the metabolic acidosis which dominates the terminal picture of fatal shock, when actually the oliguria is only a reflection of inadequate circulation to the kidneys and to the body as a whole. In such cases it is the inadequate perfusion of body tissues which produces the metabolic acidosis, and this metabolic defect in turn causes further derangements of cardiovascular function leading to progressive and even lethal circulatory failure. Preservation of satisfactory acid-base balance during the immediate post-traumatic period does not depend primarily upon the urine output, but rather on *adequate circulatory and respiratory support.*

Therefore, when one is confronted with oliguria or anuria the chief objective is to determine if the reduction in urine output is due to poor circulation and, if so, to restore adequate circulation. The first step is to be sure that arterial pressure is adequate relative to the patient's normal, for even a normal kidney cannot function without sufficient filtration pressure. Adequate arterial pressure should be attained by the use of blood volume expanders or cardiac support as outlined above. The second step is to check for and correct derangements of blood composition which can themselves suppress urine output. The CVP may then guide one in the third step of further blood volume and fluid therapy. In some patients with normal CVP the urine volume will increase following further fluid administration, suggesting that plasma volume was suboptimal, even though arterial pressure was within normal limits. An elevated and rising CVP, associated with an adequate arterial pressure, warm pink skin and normal blood chemistries, indicates that oliguria is not due to hypovolemia or dehydration. Here the blood volume is encroaching on the pumping capacity of a heart which is maintaining an adequate circulation. Fluid intake should therefore be restricted as for renal shutdown. Circulation, ventilation and nutrition should be supported to

minimize and delay the development of metabolic acidosis from renal insufficiency which may occur during the succeeding days.

The judicious use of diuretics is indicated with an increased CVP, but if the CVP is not elevated one should not force a urine output with diuretics when oliguria may be on a hypovolemic basis. In this sense, diuretics require the same thoughtful administration as do the vasopressors. Diuretic therapy should not focus attention on the volume of urine in preference to the more important consideration of adequate total circulation just as vasopressor therapy should not emphasize maintenance of arterial pressure in preference to perfusion of blood through the body tissues.

DISCUSSION

Most investigations of experimental shock have been performed with animals, and primary interest has been directed at the morbid anatomical alterations and the pathophysiology resulting from prolonged inadequate circulation leading to so-called irreversible shock. A better understanding of these pathologic alterations will undoubtedly help save more patients who have progressed to an advanced stage of shock. From a practical standpoint, however, any improvement in the clinical management of circulatory failure which can prevent the development of severe shock will be of considerably greater value in terms of the number of lives saved. Therefore, the primary clinical consideration is to avoid the conditions necessary for the development of the phenomena reported in experimental shock.

The use of measured blood volumes in the treatment of shock has definite limitations. *First*, there is a large normal variation in the blood volume of as much as 20 per cent. Measured blood volumes can be quite misleading in an emergency when one has no knowledge of an individual patient's normal volume. Using 80 ml./kg. as average, a 70-kg. man has an expected normal blood volume of 5600 ml., plus or minus 1120 ml. If his usual blood volume were 4500 ml., he could be overtransfused 1100 ml. and yet have a perfectly normal measured blood volume; he could be overtransfused 2200 ml. and still have a measured blood volume within normal limits. If his usual blood volume were 6700 ml., he could have a blood volume deficit of 1100 ml. and yet have a normal measured blood volume; he could have a blood volume deficit of 2200 ml. and still have a measured blood volume within normal limits. *Second*, even with knowledge of the patient's normal blood volume, a measured volume does not reflect how well the heart is able to handle the blood volume present at that time or how well the heart might handle any proposed increase in blood volume. *Third*, a measured blood volume becomes obsolete quickly in unstable cases where the blood volume may be

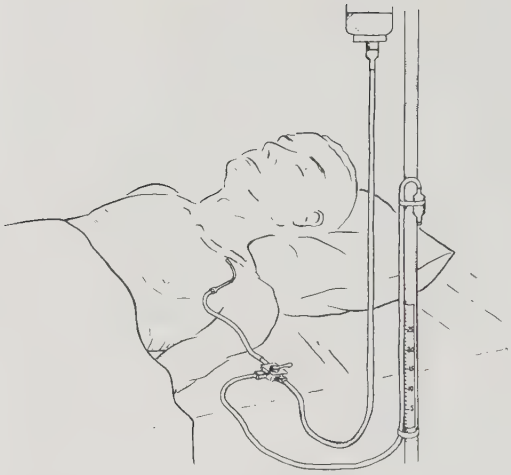

Fig. 7. Central venous pressure monitoring can be performed as a simple bedside procedure. (Wilson et al., Arch. Surg. *85:* 563, 1962.)

changing rapidly from continued depletion or blood volume replacement. *Fourth*, the number of repeat blood volume determinations that one can obtain are limited by practical, economic and safety considerations. *Finally*, reliable blood volume determinations are not available to the majority of physicians who are treating severely injured patients.

An understanding of the CVP and its relationship to cardiac pump action, blood volume and vascular tone enables one to evaluate shock or circulatory failure in terms of circulatory dynamics instead of an inanimate measured blood volume. Low venous pressure shock represents hypovolemic dynamics regardless of the measured blood volume, and responds to increased venous return. High venous pressure shock represents hypervolemic dynamics regardless of the measured blood volume, and responds to improvement in cardiac action. The CVP monitoring system can be set up quickly at the bedside with routine intravenous administration equipment available in every small hospital (Fig. 7). Serial continuous readings can be made by the physician or the nursing staff for as long a period as desired. The cost of the procedure is that of the extra intravenous tubing, and yet, with understanding, it is more valuable in the management of shock than all the blood volume measuring equipment money can buy.

One theoretical objection to using the CVP as a guide for blood volume or fluid therapy is that one might promote pulmonary edema by giving excessive fluids or volume expanders in cases of pure left ventricular failure, in which the CVP is not elevated because of a competent right ventricle. This has not occurred clinically or experimentally. Pulmonary edema from congestive failure and fluid overloading has not been observed without prior warning by an elevated CVP, indicating that cardiac failure was the predominant lesion. Pulmonary edema can de-

velop in association with hypoxia, toxemia, sepsis and inflammatory pulmonary lesions in the absence of any intravenous therapy and without an elevation of pulmonary artery pressure.[1] Pulmonary edema per se does not mean blood volume or fluid overloading. Fulminating pulmonary edema associated with hypoxia and trauma to the lung may often be controlled with tracheostomy, endobronchial suction, and oxygen administration with positive pressure ventilation, without the need or desirability for concomitant phlebotomy. Immediately following the control of pulmonary edema, persistent hypotension and oliguria associated with a low CVP have been treated successfully with plasma volume expanders. These points emphasize again the importance of respiratory function in the development and treatment of shock.

Occasionally a mechanical defect such as pulmonary or mediastinal compression will be the limiting factor in recovery, and ventilation or cardiac action can be corrected only by major surgical intervention. In such cases the circulatory dynamics must be stabilized as well as possible by optimal blood volume replacement, correction of biochemical abnormalities, and potent cardiovascular support with catecholamines while the necessary surgical correction is carried out. Such an aggressive approach has saved many patients who would certainly have died if they had been considered inoperable because of their desperate preoperative condition.

Recovery from severe circulatory failure is a process accompanied by frequent alterations in cardiovascular dynamics. Supportive therapy may therefore require frequent revisions, guided by blood chemistries and physiologic observations made at the bedside. A constant effort to maintain the blood volume in harmony with the cardiovascular capacity, together with careful consideration and correction of the specific depressants of cardiac and vascular function as they occur, will be rewarded by survival of many patients who were formerly given up as hopeless victims of "irreversible shock." Failure in therapy will then be limited to those few patients who die from resistant myocardial failure, pulmonary insufficiency or terminal renal failure — specific conditions beyond our control at present, but challenges for further investigation and advances in treatment.

CASE REPORTS

The following case reports present an interesting pair of diagnostic problems.

Case I.* A 32 year old man was hospitalized at 3:40 P.M., two hours after his chest had been crushed beneath a barricade in a go-cart accident. He complained

* Courtesy of Dr. G. B. Guarino.

of severe chest and back pain, but no abdominal pain. Examination revealed abrasions and bruises over the chest, back and extremities. The skin was ashen and clammy. The arterial pressure was 80/60, and the pulse was thready and with a rate of 115 per minute. The right chest wall was tender, and the lungs were clear to auscultation. The abdomen was soft, and there was only slight tenderness in the right upper quadrant, consistent with the bruises over the right costal margin. An intravenous infusion of norepinephrine was started to maintain arterial pressure at 100 systolic. An electrocardiogram showed sinus tachycardia and a chest x-ray revealed no evidence of intrathoracic bleeding. The hematocrit was 44 per cent.

At 11:00 P.M., the abdomen was still soft and nontender, and a repeat chest x-ray showed no abnormalities. The arterial pressure momentarily stabilized without vasopressor support, but the total urine output during the preceding 7 hours was only 60 cc. To clarify the nature of the circulatory failure, a central venous catheter was inserted by percutaneous puncture of the subclavian vein. The central venous pressure (CVP) was close to zero, confirming the suspicion of hypovolemia. After rapid administration of 500 cc. of 5 per cent albumisol, the pulse quality improved, the urine output increased, and the arterial pressure was maintained at 110/80 without further norepinephrine. The CVP remained low, so an additional 500 cc. of whole blood and 1500 cc. of intravenous fluids were given during the night. The 8-hour urine output from 11:00 P.M. to 7:00 A.M., following blood volume restoration, increased to 480 cc.

The morning after injury, the abdomen was somewhat more tender, and repeat abdominal paracentesis yielded bloody fluid from the peritoneal cavity. The CVP remained low, and the hematocrit had dropped to 37 per cent. The patient was given further whole blood replacement and taken to the operating room, where a stellate fracture of the dome of the liver was controlled with sutures and surgical packing. A large subphrenic hematoma explained the early absence of abdominal findings despite the intra-abdominal bleeding.

Comment. The absence of visible blood loss, the normal hematocrit, the lack of abdominal tenderness, the negative abdominal paracentesis, and the signs of severe chest contusion all suggested the possibility of a nonhypovolemic cause for shock. The unelevated CVP was the only *definite* finding which indicated the need for blood volume replacement.

This case illustrates the value of central venous pressure monitoring in avoiding the prolonged use of vasopressors with unrecognized hypovolemia.

CASE II.* A 3 year old boy was admitted to Denver Children's Hospital 3 days after an automobile accident in which he had suffered multiple injuries, including fractures of all 6 long bones of the lower extremities. The child had been comatose and in shock following the accident, and had received 375 cc. of blood in addition to maintenance intravenous fluids during the first 48-hour period.

On admission at 3:00 P.M. the child responded to questioning. There were lacerations of the forehead and right knee, but no signs of contusion over the chest or abdomen. The lower extremities were immobilized in bilateral traction. The arterial pressure was 106/60, and pulse was 112 per minute. During the subsequent 4 hours, the child became more lethargic and vital signs deteriorated.

* Courtesy of Dr. J. E. Strain.

By 6:00 P.M., the patient was stuporous, with neurologic signs of a brain-stem lesion. The arterial pressure was obtained with difficulty at 70/0, and the pulse was thready at 165 per minute. There was no peripheral venous filling. The lips and nail beds were pale and somewhat cyanotic. Both lungs ventilated well and were clear. The heart tones were normal, and there was no evidence of paradoxical pulse. There was a tender fullness in the right upper quadrant, and bowel sounds were absent. There was marked swelling of both thighs from the fractured femurs. The chest x-ray was normal. The hematocrit was 31 per cent.

The initial diagnosis was hypovolemic shock secondary to blood loss from the multiple long-bone fractures and possible fracture of the liver. The brain lesion appeared consistent with fat embolization. A 200-ml. infusion of 5 per cent albumin failed to improve arterial pressure or pulse quality significantly, so a central venous catheter was inserted by percutaneous subclavian vein puncture, for central venous pressure monitoring, even though hypovolemia appeared certain. Much to the surprise of the pediatric and surgical consultants, the CVP was elevated to 22.5 cm. of water. Although no cause was apparent, the diagnosis of cardiac failure was established and the child was treated with digitalization, nasal oxygen and fluid restriction. The arterial pressure improved to 90/60, but tachycardia and lethargy persisted.

From midnight to 3:00 A.M., the hourly urine output decreased from 15 cc. per hour to 8 cc. per hour, and the child's general condition appeared poor. The pulse was thready at 164 per minute, and the arterial pressure remained near 90/60. The CVP was still elevated to 16 cm., and the child's color was pale and a little dusky.

At 3:15 A.M., positive pressure ventilation with 60 per cent oxygen was started by mask. There was immediate improvement in color and vital signs, and by 6:00 A.M. the blood pressure was 108/70, the pulse was stronger and slowed to 130 per minute, and the urine volume increased to 20 ml. per hour. The CVP had decreased to 9 cm. The tender right upper quadrant mass had decreased markedly in size and was now easily recognizable as hepatomegaly. During the next few hours, the pulse slowed further to 120 per minute, and the urine volume increased to 55 cc. per hour. There was gradual mental clearing, and the positive pressure ventilation was used less frequently. An electrocardiogram that day showed marked ST segment alterations indicative of acute myocardial injury.

That evening, 30 hours following admission, the CVP was down to 10 cm. and there was a good diuresis. The hematocrit was 26 per cent. Blood was given slowly throughout the night. The following morning the vital signs were stable, but the CVP had increased to 16 cm. and the urine output was down. Further treatment of the anemia with whole blood was withheld, and diuretics were administered with good result. By that evening the CVP had decreased to 11 cm. and the vital signs were stable.

Five days after admission, the electrocardiographic evidence of myocardial injury had resolved, the child was alert and responsive, and the hematocrit had risen to 44 per cent. Subsequent recovery was uneventful.

Comment. Cardiac failure was entirely unsuspected after careful evaluation by resident staff and several attending physicians. The elevated CVP alone established the diagnosis of nonhypovolemic circulatory failure and guided therapy during recovery. The case illustrates the important role of respiratory support in the treatment of circulatory failure, and further illustrates how easily the need for mechanical

ventilatory support can be overlooked. In this case hypoventilation was probably secondary to the brain injury.

SUMMARY

Early and aggressive treatment of acute circulatory failure is a keystone in the overall management of severe trauma. Successful restoration and maintenance of an adequate circulation requires a coordinated support of cardiac pump action, circulating blood volume, and vascular tone.

Continuous central venous pressure monitoring reflects the minute-to-minute relationship between blood volume and cardiovascular capacity, and thus permits integration of optimal blood volume maintenance with support of cardiovascular function.

An outline for the management of severe circulatory failure is presented. The importance of respiratory support is emphasized, and the significance of the urine output, vasopressor therapy and the choice of blood volume expanders is discussed.

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Parenteral Fluid Therapy in the Injured Child

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Parenteral fluid therapy for the severely injured child is a problem peculiar to pediatric surgery, and methods used in the treatment of adults must be modified. Children, and particularly infants, are not small adults, and the differences between the child and his elders are most apparent in the realm of fluid and electrolyte balance.

The purposes of this paper are to underscore some of these differences, to point out some of the difficulties in fluid management of severely injured children, and to present a practical approach to planning parenteral fluid therapy for the injured child.

TECHNIQUES OF PARENTERAL FLUID THERAPY

Techniques of administration of parenteral fluids present the first problem of the surgeon treating the injured child. The younger child has small easily injured veins, buried under a generous layer of fat. The use of hypodermoclysis, rectal instillation or bone-marrow infusion to supply fluids has been strongly criticized.[22, 26] The intravenous route offers the safest, quickest and most accurate control over fluid management. The veins of the dorsum of the hand in children, or the veins of the scalp in infants, are best used for this purpose.

Rapid access to a vein is often imperative; this has been best achieved by cannulation of a large and readily accessible vein with a plastic catheter. The most reliable veins for such "cut-downs" are the long saphenous vein at the ankle, the cephalic or basilic vein in the antecubital space, and the external jugular vein in the neck.[9, 19, 22]

The intravenous needle or cannula must be carefully secured and the child must be restrained so as to thwart his efforts to remove it; even the tiniest infants are expert at removing "cut-downs" quickly and completely, if given the chance.

Particularly in the smaller child, the equipment through which the fluids are delivered is important. The use of a bottle of intravenous fluid with too large a capacity is hazardous. Should fluid be inadvertently administered rapidly, and such a bottle allowed to empty itself, the patient would soon be overloaded. The use of containers especially designed for small patients would obviate this hazard.

PLANNING OF PARENTERAL FLUID THERAPY

It will be apparent that the plans for fluid therapy presented below usually tend toward undertreatment. It is preferable to keep the patient on the dry side; although small children and infants are able to handle surprisingly large water loads,[14] errors of underhydration are more easily corrected than are those of overhydration.

The purpose of therapy can be subdivided into three parts:[8]

1. Provision of maintenance requirements,
2. Repair of pre-existing deficits, and
3. Replacement of continuing abnormal losses.

Estimating the fluid and electrolyte requirements for each individually, one can then add them to determine the total fluid and electrolyte to be provided in a given period of time.

MAINTENANCE REQUIREMENTS

Estimates of the maintenance requirements for water, sodium, potassium, and nitrogen among normal children of various ages[1, 8, 11, 12, 17, 24, 25] have been modified as shown in Table 1, in keeping with the desire for slight undertreatment.

Table 1. Maintenance Requirements

	NEWBORN	OLDER INFANTS * (Less Than 1 Year)	OLDER CHILDREN (Over 1 Year)
Water	25 ml./kg./24 hrs.	50 ml./kg./24 hrs.	25–50 ml./kg./24 hrs.†
Sodium	17 mEq./liter of water intake	35 mEq./liter of water intake	35 mEq./liter of water intake
Potassium	None	35 mEq./liter of water intake	35 mEq./liter of water intake

* The estimates in the groups less than 1 year of age are based on infants nursed in a high-humidity environment.

† The larger the child, the smaller the estimate.

Protein must be provided for infants undergoing prolonged parenteral fluid therapy. The child is able to incorporate this rapidly into his metabolic pool.[21] Albumin is usually administered at the rate of 1.25 gm./kg./24 hours.

REPAIR OF PRE-EXISTING DEFICITS

Estimates of pre-existing deficits are most inaccurate, and it is here that one must proceed most cautiously. Laboratory measurements should be used only in conjunction with the history and clinical appearance of the patient.

Table 2. Fluid Compartments Expressed as Percentage of Body Weight

	NEWBORN	INFANTS (LESS THAN 1 YEAR)	1–5 YEARS	OVER 5 YEARS
	%	%	%	%
Blood volume	9	8	8	7
Extracellular water	40	30	25	20
Intracellular water	35	40	40	45
Total body water	75	70	65	65

Published data on the various fluid compartments in children[3, 4, 7, 20, 23, 24] have been adapted as shown in Table 2.

The rate at which repair is to be accomplished is as important as the amount of repair solution to be given and depends on the condition of the patient and the urgency of the planned surgery.

Three examples of the types of pre-existing fluid and electrolyte deficits most frequently encountered among injured children follow.

Acute Hemorrhage

Children vary widely in their responses to acute hemorrhage. Some show intense vasoconstriction after minimal blood loss; others maintain vital signs and a near-normal clinical appearance after surprisingly large amounts of blood loss, only to develop profound shock quite suddenly. Whole blood transfusions up to 25 ml./kg. may be administered rapidly to patients in shock from acute hemorrhage. Central venous pressure[27] as well as pulse rate and arterial pressure should be carefully monitored to judge the patient's response.

One method of estimating pre-existing blood volume deficit is by measurement of plasma volume, using I^{131}-tagged albumin or Evans blue dye. This estimate can then be compared with the patient's predicted plasma volume, based on his weight before injury, to determine his current deficit.

Operative blood loss may be determined by measurement of blood on drapes, sponges and in suction bottles; by accurate weighing of the patient immediately preoperatively and postoperatively; or by serial measurements of the patient's plasma volume with I^{131}-tagged albumin or Evans blue dye.

Burns

Estimates of burn fluid loss may be obtained by various means. Fluid loss in a patient who has suffered a burn involving less than 25 per cent of the body surface area is seldom a serious problem. Most workers agree that fluid losses are large and relatively constant in patients with second or third degree burns involving more than 50 per cent of the skin. The

area of disagreement centers around the methods of estimating burn fluid loss in patients suffering from second or third degree burns involving 25 to 50 per cent of the surface area. In general, two types of approach to the problem have been used.

Moore[15] postulates that during the first 48 hours the patient will lose a fixed proportion of his extracellular fluid into the burned area, regardless of the exact extent of the surface burned. Applied to children, this method calls for the administration in the first 48 hours of a volume of colloid-containing isotonic saline solution equal to 12 per cent of the body weight, half of this being given during the first 12 hours after the burn. In addition, Moore advises giving normal saline, the amount depending on the nature of the burned surface—the drier the burn, the less the extra saline requirement.

Evans' formula[6] assumes that the patient with second or third degree burns will lose an amount of fluid proportional to the surface area of the burn. A modification of this formula, which has worked well with children, calls for the administration of 1.0 ml. of plasma (or, better yet, 5 per cent albumin in buffered saline) and 0.5 ml. of normal saline per 1 per cent second or third degree burn per kilogram of weight in the first 24 hours, half of this ration being given in the first eight hours. Burns of greater than 50 per cent of the body surface are treated as if they were of 50 per cent area. The percentage of body surface burned may be estimated by Lund and Browder's[13] data on skin surface area (see Table 3).

Table 3. Approximate Percentage of Body Surface

	NEWBORN	3 YEARS	6 YEARS	12 YEARS
	%	%	%	%
Head	18	15	12	6
Trunk	40	40	40	38
Arms	16	16	16	18
Legs	26	29	32	38

As an example of the application of these two methods, the following hypothetical case is presented:

An 18 month old child weighing 10 kg. enters the hospital shortly after sustaining second-degree burns of the lower trunk and legs estimated at 30 per cent of his body surface area. The estimate of burn fluid loss would be made as follows:

1. By Moore's method:

Colloid requirement for 48 hours = 10 kg. × 12% = 1200 ml.
Colloid requirement for first 12 hours = 600 ml. or 50 ml./hr.

2. By modification of Evans' method:

Colloid requirement (24 hours) = 1.0 × 30% × 10 kg. = 300 ml.
Saline requirement (24 hours) = 0.5 × 30% × 10 kg. = 150 ml.

3. To these estimates must be added the fluid and electrolytes necessary for maintenance, as explained in the previous section (Table 1):

Water 50 × 10 kg. = 500 ml./24 hours
Na 35 × 500 ml./1000 ml. = 17.5 mEq./24 hours
K None would be given to a burned patient at this phase

These needs would be met by approximately 20 ml./hr. of an 0.2 per cent NaCl solution.

4. Thus, the initial fluid estimate for such a patient would be:

	MOORE'S METHOD	MODIFIED EVANS' METHOD
Colloid	50 ml./hr.	20 ml./hr.
Normal Saline	As necessary to compensate for for skin water and sodium losses.	10 ml./hr.
0.2% NaCl	20 ml./hr.	20 ml./hr.
Total Fluid	70 + ml./hr. for 12 hrs.	50 ml./hr. for 8 hrs.

5. With deep burns and in small children, administration of whole blood will be necessary to compensate for red cell loss.

A word of caution is in order in the application of a formula to the individual case. Overhydration is a real danger in these patients, particularly if there has been respiratory burn. Since there is wide variation in requirements calculated by the different methods, and since considerable error may enter into the estimate of percentage of body surface burned, the chosen formula should be used only as a starting point. The patient must be monitored closely for several hours; the formula should then be discarded and the patient should be given the *minimum amount of fluid* necessary to maintain adequate vital signs, hematocrit, and urinary output with stable urinary specific gravity.

Dehydration

Except under experimental conditions, water loss is never found without some concomitant loss of electrolytes. Whether electrolyte loss is disproportionate to water loss can often be surmised from the history and physical examination. The history and the clinical appearance of the patient are the bases for estimation of water loss; for approximating electrolyte loss one can use the comparison of the patient's serum electrolyte values with normal values.

1. Water Loss. Deficits in total body water are extremely difficult to determine with any degree of accuracy. It has been estimated that the signs of dehydration are not clinically detectable until approximately 6 per cent of total body water has been lost. Severely dehydrated

children may have deficits up to 25 per cent of total body water. These limits are used as guide lines in planning fluid therapy in such patients.

2. ELECTROLYTE LOSS. "Normal values" for electrolytes at this institution[18] are shown in Table 4.

Table 4. Normal Values for Serum Electrolytes

Na	136–143 mEq./L.
Cl	97–104 mEq./L.
K	4.1–5.6 mEq./L. (up to 7.0 mEq./L. in the newborn)[16]
Total CO_2	18–23 millimols./L.

Sodium administered to a sodium-depleted patient is not only distributed throughout the extracellular space, but also moves into the cells. The quantitative aspects of this shift are unpredictable, but can be minimized by slow, gradual repair and by the simultaneous supplying of potassium. Thus, a first approximation of the amount of sodium required to raise the serum level to 140 mEq./L. in a hyponatremic patient may be derived from the formula:

$$Na^+ \text{ required} = (140 \text{ minus observed } Na^+ \text{ level}) \times (\text{extracellular water})$$

This estimate can also be made by assuming that sodium thus administered is distributed throughout the total body water, calculating the sodium required based on the plasma sodium deficit in mEq./L. times total body water, and then giving one-half the calculated amount as an initial dose.[1]

Either method accomplishes *partial* restoration of the cation concentration in the body water and return of the osmolar concentration of the body water *toward* normal. It is neither necessary nor wise to correct sodium deficits completely in a short period of time.[25] It is emphasized that sodium is important in relation to potassium and water—a low serum sodium does not necessarily indicate a sodium deficit; instead, it may represent the result of a potassium deficit or of a surfeit of water.

Sodium is usually given as sodium chloride, but if there is also a marked acidosis some may be given as sodium bicarbonate. A set of calculations similar to those in the paragraph above will give an initial estimate of the amount of bicarbonate to be given to a patient with metabolic acidosis.

In alkalotic patients depleted of chloride similar calculations can be made, and the chloride may be supplied as sodium chloride. If severe alkalosis is present, ammonium chloride may be given.

Potassium deficits cannot be estimated from a knowledge of the serum level. Such deficits are usually approximated from a knowledge of the type and amount of fluid lost and its expected potassium concentration.

Replacement of Continuing Abnormal Losses

Abnormal losses often continue during therapy. In patients suffering from blood loss or burns, these continuing losses are best estimated by close monitoring of the patient. In patients losing fluid from the gastrointestinal tract, it becomes extremely important to measure these losses. Gutierez, Sukarochana and Kiesewetter,[10] studying 30 infants and children who were on gastric suction postoperatively, found average electrolyte composition of the gastric aspirate as given in Table 5. There was

Table 5. Approximate Electrolyte Composition of Gastric Aspirate

AGE	Na (mEq./L.)	K (mEq./L.)	(Cl (mEq./L.)
Less than 6 months	53	11	80
Over 6 months	89	14	104

wide variation, however, and therefore actual measurement of the electrolyte composition of the fluid and its accurate replacement are in order if these fluid losses are great or if the patient is severely depleted.

INITIAL ESTIMATE OF FLUID AND ELECTROLYTE REQUIREMENTS

Separate estimates have now been obtained of fluid necessary for (*a*) maintenance, (*b*) repair of pre-existing deficits, and (*c*) replacement of continuing abnormal losses. Having determined the rate at which correction of any pre-existing deficit should proceed, one can then add these three portions and arrive at an initial estimate of the patient's fluid and electrolyte requirements. If the various solutions are miscible, it is preferable to mix them together and administer them as a "cocktail." For the patient in shock due to decreased vascular volume, from whatever cause, the initial resuscitative efforts should be directed toward restoring blood volume, using blood or colloid as appropriate, in accordance with Moore's[15] advice.

MONITORING THE PATIENT

In treating injured children, initial estimates of the patient's fluid and electrolyte requirements are made as outlined above. No matter how exact these estimates seem, or how erudite the reasoning by which they have been derived, it must always be remembered that these are only estimates; they must be checked continually against the patient's responses. In clinical practice there is usually a wide margin of safety

within which the patient can protect himself from inaccuracy of the estimate, but when the patient's homeostatic ability is weakened by injury or disease or when, as in the cases of many small children and infants, his metabolic responses are subject to wide, poorly understood shifts,[2, 5, 21, 26] the margin of safety narrows. It then becomes necessary to follow the patient's response to therapy closely, and to check on the accuracy of the estimates frequently and thoroughly.

Which parameters of the patient's response should be followed and how frequently depends, of course, on the severity of the patient's condition. Intake and output by various routes must be accurately measured. Urinary output and specific gravity are among the best indications of the patient's response to therapy. The patient's weight should be determined at least once a day, especially in small infants; rapid fluid shifts are often first detected by changes in body weight. It is possible to measure serum electrolyte levels, serum protein levels, and hematocrit repeatedly, even in small infants, using micro- and ultramicro laboratory techniques. In severely depleted children, knowledge of electrolyte losses in urine or gastrointestinal drainage is invaluable in planning fluid therapy.

Most important, however, are frequent review of these data by the surgical staff and periodic adjustments in the fluid and electrolyte program. Especially in small children and in infants, the patient's response should be reviewed several times a day. Fluid orders are written for periods of only four to eight hours and reviewed periodically; parenteral intake is reordered as indicated. The response of critically ill infants should be evaluated even more frequently than this.

CONCLUSIONS

At our current level of understanding of the various fluid and electrolyte shifts taking place in injured children, the details of these responses to injuries and to our attempts at replacement are extremely complex. What has been presented here is not a theoretical discussion of these responses as we know them, but a practical approach to an initial estimate of fluid and electrolyte requirements and to subsequent adaptation of treatment to the patient's response.

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Late Care of Burns

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After the first few days, during which the circulating blood volume and the electrolyte balance are restored and maintained, the care of the patient with a major burn becomes one of controlling infection and restoring surface coverage. These two are closely related, and one cannot be achieved without attaining the other.

In the second-degree burn, healing will follow if infection does not occur. Therefore, a reverse type of sterile technique and isolation is required, with care being taken to prevent the doctors, the nurses and the medical personnel from contaminating the wound. This healing would occur in 14 to 21 days, and a spontaneous separation of the flat thin eschar should appear.

In deep dermal burns, the "be-darned burns"—"darned if you do and darned if you don't debride"—there is a paucity of epithelial elements to initiate wound coverage and infection, and slow healing results in dense hypertrophic scarring. If clean, these burns may heal spontaneously in 30 to 40 days, but in many cases debridement and skin grafting will result in a more rapid recovery with a better functional and cosmetic result.

In the third-degree burn with the depressed eschar and thrombosed vessels, "like leafless branches after a winter frost," debridement becomes a necessary procedure if infection is to be controlled and the area is to be skin-grafted. Debridement can be carried out by the simple mechanics of repeated dressing changes, since all burns, no matter what the type of initial care (Vaseline gauze pressure dressing or the exposure method), are now being dressed with bland ointments, antibiotic ointments, or various antibiotic, antiseptic or electrolyte solutions. Other methods of debridement are by chemical agents (phosphoric or pyruvic acid) or enzymatic agents.[1] These methods are still experimental, and they cannot yet be recommended for general usage in major burns. The best method of debriding the burn wound is sharp surgical dissection with the patient under a light general anesthetic.

The recent trend to earlier and earlier debridement has much to recommend its continuation; it prevents infection, allows early skin grafting, reduces hospital stay, and finally gives less post-burn deformity and contracture. Of course, there are two essential factors: the burn

must be third degree, and debridement with its hemorrhage and necessary anesthetic must not jeopardize the life of the patient.

This early debridement (one to seven days) works well in deep burns of small areas, such as the backs of the hands, but debridement (necrectomy) and immediate split skin grafting of major burns, 30 per cent and upward, must for the time being be considered experimental and be done only in major centers where all care and facilities are available.

Recently, a method for debridement by abrasion was reported.[2] This method may have much to offer if it is truly possible to remove all tissue rapidly with a minimum of damage to underlying viable tissue.

In many major burns, after debridement has been accomplished there is not enough skin available to give coverage even if this skin is used as small rectangular grafts (so-called "postage stamp grafts"). In such cases, many lives have been saved by the use of homologous skin (cadaver or donor), which will give a clean closed wound for from 10 to 120 days and allow a vigorous attempt to get the patient in good enough general physical condition so that his own limited donor skin can be made available or reharvested if necessary. During this period, general vigorous supportive care should be instituted.

This general supportive care should maintain nutrition at the highest possible level, combat anemia, combat infection and give psychological support. A daily diet of 2 to 3 grams of protein per kilogram of body weight, with at least 30 calories of carbohydrate to each gram of protein, should be maintained. This may require as high as 5000 to 6000 calories daily. Body weight is a good check on the general nutritional state. Multiple vitamins are necessary, with a daily intake of at least 1 gram of vitamin C. The seriously burned patient develops severe anemia and hypoproteinemia, and repeated whole blood transfusions are necessary during the entire hospital stay.

Antibiotics are of greatest importance when there is evidence of bacteremia, and a blood culture with bacterial growth helps in the selection of the indicated antibiotic.

Priority for skin grafting should be assigned to areas of functional or cosmetic importance, such as hands, face and flexion areas.

Intermediate thickness split skin grafts (0.010 to 0.016 inch) should be used, because these leave a donor area which may be reharvested if necessary and also give a good functional and cosmetic result to the recipient areas. It must be remembered that skin varies in thickness in different body areas and with different age groups. Surely the use of the electric dermatome which takes grafts quickly from large areas at a uniform depth has saved many lives. The skin graft donor area is usually covered with a nonadherent, nonocclusive pressure dressing, and should not be changed until healed unless pain or exudate indicates infection.

In major burns it is usually not necessary to sew the graft in place,

except on face or hands, especially if the grafts are backed with nonadherent mesh or gauze.

The recipient area should be examined in about five days, and thereafter about every other day, with reapplication of a nonadherent, near-dry dressing, or wet (not saturated) medicated dressing. If by clinical evaluation, pain, temperature, etc., it is felt that infection is occurring in the recipient area, dressings should be changed earlier (in three to four days) and a vigorous regimen of moist antibiotic antiseptic (Furacin, Betadine) dressing changes instituted. In only a few areas (perineum, genitalia and possibly face) is the use of the exposed graft occasionally indicated.

The most important single item in late burn care is a vigorous, time-consuming, undramatic obsession with keeping the area clean, changing the dressings and skin grafting as early as possible. This is the duty of the physician who assumes the responsibility of total care of the severely burned patient, and should not be delegated to the inexperienced physician or registered nurse. Burn care is hard work, and the best result that can be achieved leaves much to be desired; deformity and cosmetic and functional deficits follow in the wake of the burn. Physiotherapy, proper splinting, and mobilization of extremities will do much to maintain function. Many specialized procedures, such as arthrodesis of finger joints and tarsorrhaphy of eyelids, can be used to prevent disability, restore loss of motion and give better skin coverage in critical areas.

In the future it is hoped that all patients with major burns will be rapidly transported to major burn care centers where advanced medical and surgical resources are available to restore these victims of the most catastrophic of all injuries.

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The Diagnosis and Treatment of Gas Gangrene

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GAS GANGRENE as a complication of trauma occasionally occurs when prophylaxis is too little and invariably kills when therapy is too late. If delayed until the complete clinical picture becomes manifest, attempts at treatment are frequently futile. Prompt and extensive surgery is the only effective measure available for the therapy of this disease, and while antibiotics and antisera may augment debridement, these agents by themselves are inadequate. It becomes important then that diagnosis be both early for treatment to be successful and accurate to spare those with similar but more benign infections the operative procedures that gas gangrene requires.

MICROBIOLOGY

Three groups of clostridia are found in association with traumatic wounds. The first, including Clostridium perfringens (welchii), Cl. novyi (oedematiens), and Cl. septicum, are toxigenic and proteolytic organisms capable of causing the clinical syndrome of gas gangrene. Of these, Cl. perfringens is the most frequent, occurring in 56 to 80 per cent of cases,[9, 10, 19] although in the majority of wounds containing clostridia more than one species of these bacteria are found.[15] The second group, including Cl. sporogenes, Cl. histolyticum, Cl. bifermentans and Cl. fallax, are nontoxigenic, but because of their proteolytic enzymes augment the infection by supplying additional nutrients for bacterial growth.[15] The third group includes nontoxigenic and nonproteolytic strains such as Cl. butyricum and Cl. tertium, which appear only as contaminants in these wounds.

The clostridia are widely distributed in nature, occurring in both man and animals as well as in the soil. Clostridium perfringens and Cl. sporogenes are uniformly present in the intestinal tract of man, while other species of clinical importance such as Cl. novyi and Cl. septicum are frequently but not invariably present.[6, 11, 12, 22, 23] Clostridium perfringens has been found both as spores and as vegetative forms in soil

free from known fecal contamination, suggesting that these organisms can proliferate in this environment as well as in the gut.

These organisms are obligate anaerobes and cannot multiply in the relatively high oxidation-reduction potential that obtains in healthy muscle tissue. In the event of direct injury or local circulatory embarrassment, however, there is a lowering of the redox potential, and of the pH, both factors tending to produce an environment suitable for clostridial growth.[4, 19] Once multiplication has been initiated, toxins produced by these bacteria cause death of adjacent previously healthy muscle with progressive extension of the gangrenous process.

The exotoxins (extracellular enzymes) elaborated by the clostridia, rather than any invasive capacity of these organisms themselves, are responsible for gas gangrene, although there is not a direct correlation between *in vitro* toxin production and pathogenicity.[19] The alpha toxin, a lecithinase, produces hemolysis followed by anemia, hemoglobinuria, oliguria and jaundice. The theta toxin, a second toxin with similar enzymatic activity, is produced to a lesser extent. The upsilon toxin, a hyaluronidase, causes dissolution of the intracellular matrix, thereby permitting wider dissemination of the infectious process. In addition, other enzymes formed by these bacteria cause hydrolysis of proteins, lipids and nucleic acids as well as necrosis of tissue.[16] The activity of several proteolytic and saccharolytic enzymes results in the tissue crepitus which characterizes gas gangrene.

CLINICAL FEATURES

It is important to recognize that the finding of clostridia in a wound does not establish the diagnosis of gas gangrene. On the contrary, contamination of wounds with these anaerobes is quite frequent in contrast to the relative rarity of gas gangrene.[3]

Clostridia may be present in traumatic wounds under three circumstances: (1) simple contamination, (2) anaerobic cellulitis, and (3) gas gangrene (clostridial myositis or myonecrosis). Simple contamination of war wounds with these organisms in the absence of significant infection was found to occur in 18 to 46 per cent of cases.[9, 14] If proteolytic clostridia are present they may proliferate on any dead tissue, blood clots or purulent material present in the wound without, however, invading viable tissue. A thin, brown, foul-smelling seropurulent discharge occurs, and the deeper parts of the wound develop a greenish-black appearance. The clostridia usually disappear from these wounds without specific therapy, although if staphylococci or Cl. perfringens is present there may be some delay in wound healing.[25]

Anaerobic cellulitis develops in about 5 per cent of traumatic wounds containing clostridia.[9] Following an incubation period of three to four

days, multiplication of these bacteria occurs within the wound and connective tissue spaces leading from the injury site. A moderate brown foul seropurulent discharge results with, however, little or no local pain, edema or discoloration of the skin. Gas formation may be marked, and this also is confined to the fascial planes and never involves the muscle. Manifestations of systemic reaction, such as fever, tachycardia or toxemia, are characteristically minimal.

Gas gangrene was noted to develop in 1.5 per cent of war wounds containing anaerobes.[9] The incubation period is usually short, ranging from seven hours to three and one-half days in Cl. perfringens infections, although it may be as long as six weeks after injury in the case of Cl. novyi. The earliest symptom is a feeling of tightness or weight in the affected area, followed by the sudden onset of severe pain. Mental confusion and toxemia follow, and there is a tachycardia often out of proportion to the moderate temperature elevation. Local swelling and edema rapidly occur, and the skin, which was at first white, becomes reddened and discolored and later sloughs. A thin, watery, usually foul discharge appears and soon becomes serosanguinous and profuse. The involved muscle is friable, purplish-black in color, and loses its contractile response to stimulation. Gas may be present in the infected muscle, but this often is not apparent until late in the course of the disease. Hemolytic anemia rapidly develops, followed by hemoglobinuria, oliguria, shock and jaundice; death may occur as soon as 24 hours following the onset of symptoms.[3]

Although it is unknown why only a small percentage of all wounds containing clostridia progress to gas gangrene, certain factors are recognized that predispose to this complication. In four cases of gas gangrene following operation, all of the patients were being treated for ischemic vascular disease in the affected limb, and in addition two of them were diabetic.[3] The incidence of gas gangrene also appears to be related to the lapse of time between wounding and surgical treatment. Eight cases per thousand occurred in wounds debrided one day after injury, while almost six times this number occurred in wounds treated 3.75 days after injury.[7] Finally, the site of injury seems to be of importance; gas gangrene was three to five times more frequent in wounds of the thigh and buttock than in wounds of the shoulder, arm or distal lower extremity.[7, 25]

DIAGNOSIS

The diagnosis of gas gangrene must be primarily on clinical grounds in order that this rapidly fatal infection may be arrested. The acute onset of pain at the site of injury, in association with fever, mental confusion and toxemia, should suggest this diagnosis.[3] Although a "mousy" or

"rotten meat" odor and crepitus are described as characteristic of gas gangrene wounds, these features are often absent until relatively late in the disease.[7]

Certain laboratory tests are of some value in supporting this clinical diagnosis. Radiologic examination of the affected area may demonstrate gas in the tissues which occasionally cannot be appreciated by physical examination.[3]

Gram-stained smears of the infected tissues will in the case of gas gangrene reveal many large gram-positive bacterial rods and small numbers of degenerated leukocytes. Cultures of these wounds yield clostridia, and blood cultures may also be positive, although these confirmatory data will be available only after appropriate therapy has been instituted.[22, 26]

NONCLOSTRIDIAL INFECTIONS SIMULATING GAS GANGRENE

It has been emphasized that the finding of clostridia in a wound does not establish the diagnosis of gas gangrene. Further, it is important to recognize that not all wounds showing gas formation are infected with clostridia.

Gram-negative bacteria can produce crepitant abscesses in patients with diseases such as diabetes and leukemia who have decreased resistance to infection. Blum (1961) described a klebsiella abscess in a diabetic patient in which gas formation was apparent both by physical and by radiological examination.[1] Wills and Reece (1960) reported four cases of "nonclostridial crepitant cellulitis" in diabetics who manifested both neuropathy and peripheral vascular disease.[24] These infections were caused by a variety of streptococci and enteric gram-negative bacteria. Clinical differentiation from clostridial myositis was on the bases of an insidious onset of pain, a slow course, the presence of gas only in the fascial planes, and the absence of necrotic muscle.

A true myositis, however, may be caused by the combination of anaerobic streptococci with either Streptococcus pyogenes or Staphylococcus aureus.[9] Following an incubation period of three to four days, there is a gradual onset of fever, toxemia and tachycardia. Local pain is variable, but it can be quite severe. The wound appears wet and edematous, although odor and gas formation usually are not marked. A large amount of thin, blood-stained, seropurulent fluid exudes from the wound, which on gram-stained smear shows few bacteria but large numbers of leukocytes. Shock is not as prominent as in clostridial myositis, and the course is less fulminant; however, five of 19 cases reported by MacLennan (1943) were fatal.[9]

The therapy for these infections consists of appropriate antibiotics and adequate local drainage.

PROPHYLAXIS AND THERAPY

The measures available for the prophylaxis and therapy of gas gangrene are primarily surgery and secondarily antibiotics, antitoxin and general supportive care. As pointed out above, adequate debridement with removal of all compromised tissue prevents the growth of clostridia and thus the development of gangrene.

Simple contamination of wounds with clostridia requires no specific treatment beyond adequate surgical attention.[9] In the event of delayed wound healing associated with Cl. perfringens and Staph. aureus, the use of antibiotics seems justified. The prophylactic use of antibiotics has been suggested for high-risk patients such as those with diabetes and occlusive arterial disease. Gye et al. (1961) reported four cases of gas gangrene among five patients who had surgery for ischemic vascular disease.[3] It has been pointed out, however, that clostridial growth is initiated in areas devoid of circulation, and thus antibiotics can reach these contaminated areas only by the relatively inefficient mechanism of diffusion.[19]

Anaerobic cellulitis may be treated adequately by surgery alone, which should provide relief of local tension, removal of necrotic tissue, and adequate drainage.[9, 25] Antibiotics are probably useful in these cases, especially if there is superimposed infection with streptococci or staphylococci. Antitoxin is not required here, and this infection does not progress to clostridial myositis.[9, 25]

Gas gangrene must be treated first by prompt and complete debridement or amputation if necessary, and it is uniformly agreed that in the absence of adequate surgery all other therapeutic measures are valueless.[3, 7, 9, 10] Gye et al. (1961) expressed the clinical impression that antibiotics are a useful adjunct in the treatment of gas gangrene.[3] Langley and Winkelstein (1945) emphasized that, while antibiotics may be useful after debridement, they do not prevent the spread of gas gangrene during the preoperative period.[7]

Of the antibiotics available for the prophylaxis and therapy of clostridial infections, penicillin and the tetracyclines are the most effective. Nagler (1945) found that all of 44 strains of Cl. perfringens were inhibited by 0.2 units/ml. of penicillin,[13] while Newton et al. (1955) observed that a few strains of Cl. novyi and Cl. sporogenes required up to 6.4 units/ml. for inhibition.[14] All strains tested were sensitive to 0.8 mcg./ml. of chlortetracycline, although some were resistant to chloramphenicol, requiring up to 32 mcg./ml. for inhibition.[14] Antibiotic therapy then should consist of 10 to 20 million units daily of parenteral penicillin or 2 to 4 grams daily of an intravenous tetracycline.

Antitoxin has been part of the therapeutic program in most of the reported cases of gas gangrene.[3, 7, 9, 17, 25] MacLennan (1943) reports that

survival rates in his patients increased from 20 per cent to 75 per cent when serum was used.[9] Further, certain animal experiments suggest a synergistic effect of combined antibiotic-antitoxin therapy,[5, 13, 21] although this result is not invariable[8] and has not been confirmed in clinical studies.[8, 10, 25] Gye et al. (1961) stated that antitoxin was of "questionable value," but that it should be used in the therapy of this disease.[3] In contrast, MacLennan and Macfarlane (1945) were able to detect excess administered antitoxin in the serum in nine fatal cases as long as 96 hours before death. It was noted, however, that surgery was probably inadequate in all nine of these cases.[10] Newton et al. (1955) did not list antitoxin as a factor in controlling gas gangrene during the Korean War,[14] and Lindsay et al. (1958) reported that the U. S. Army had dropped gas gangrene antitoxin from its standard stock list. Further, the use of antiserum is in itself not an innocuous procedure. In a series of 82 treated patients, 75 per cent demonstrated some form of hypersensitivity to the serum, ranging from urticaria and fever to fatal anaphylaxis in one case.[7]

The recommended therapeutic dose of polyvalent gas gangrene antitoxin varies from 40,000 to 60,000 units. This is repeated every four to six hours until clinical improvement occurs, usually within a few days.[7, 9, 17, 25] The antitoxin is first given intravenously, as it has been found that after intramuscular administration detectable circulating levels can be found only after a lag period of 12 to 24 hours. Patients should be skin tested for hypersensitivity before administration of the antiserum, and desensitized if necessary.[7]

General supportive care is also of importance in the treatment of gas gangrene. Blood transfusions should be given to control anemia, and electrolyte abnormalities (usually acidosis) should be corrected. Fluid restriction may become necessary if oliguria develops, and steroid therapy has been recommended in the event of shock.[26]

Despite the many recent advances in chemotherapy of infectious diseases, the admonition of Langley and Winkelstein,[7] written 17 years ago, remains valid today: "We have confirmed, and it is not possible to overemphasize the fact, that early surgical debridement is the one single measure of vital importance in the prophylaxis as well as the treatment of gas gangrene."

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The Problem of Frostbite in Civilian Medical Practice

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On the pass of Mt. Cenis, between France and Italy, is the "Chapelle des Transis," a refuge dedicated many centuries ago to travelers frozen on their journeys through the Alps. Technological improvements have since reduced the hazards of winter life, but cold injury is still an important medical problem particularly in wartime, where under field conditions disability has often reached epidemic proportions.[38]

A review of 54 cases of frostbite treated during the past five years at the Colorado General and the Denver Veterans Administration Hospitals has convinced us that cold injury is an important medical problem within the civilian population as well. Hospitalization and rehabilitation constituted a heavy financial burden to the individual, his family and the community. Crippling amputations or late neurovascular complications often resulted.

The cases exhibited a wide spectrum of clinical manifestations ranging from mild erythema to totally destroyed extremities. It was apparent from the lack of uniformity in treatment regimens that no one mode of therapy has been accepted as entirely adequate. This fact emphasizes the lack of general agreement in many facets of the etiology, pathophysiology and proper care of this form of trauma.

Although no new basic information can be gained from a review of

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cases such as this, it does provide a convenient background to illustrate the complexities of the problem as it now exists in our civilian hospitals. At the same time, many aspects which require further concerted clinical and laboratory investigation under rigidly controlled conditions will become obvious. Various questions will be raised, many of which cannot be answered with certainty at the present time.

NOMENCLATURE AND CLASSIFICATION

What Is Cold Injury?

Cold injury is a composite term applied to a variety of hypothermic traumas which result in similar clinical and pathologic manifestations. The principal environmental factors leading to tissue injury are wetness and cold. Names given to the subdivisions of cold injury are arbitrary and have evolved on the basis of the circumstances of injury. Thus, frostbite is caused by exposure to freezing cold. The combination of nonfreezing cold and wetness produces trench foot, shelter foot and immersion foot.

The extent to which actual tissue freezing occurs in frostbite is controversial,[32] except for the special circumstances of "high altitude frostbite"[8] where intensely cold dry air freezes tissues within a few seconds. The usual victim of frostbite is not seen by a physician until long after the injured member has been thawed. In the present series, the shortest time between injury and medical examination was 30 minutes, but the average time was five and a half days. Commonly, the history is inadequate to determine whether solidification of the member has occurred. Occasionally, a patient will arrive at the hospital with actual tissue freezing still demonstrable.

Case I (CGH, 164687). A 27 year old man was driving while intoxicated. His car ran out of gas and he walked toward a nearby farmhouse, became tired and lay in a snowdrift to rest. He was found 7 or 8 hours later and brought immediately to the emergency room. Both hands and both feet as well as the ear and nose tips were frozen solid. He was admitted to the ward and allowed to warm in bed at room temperature which is kept at about 72° F. After thawing, which required several hours, the distal extremities were white (Fig. 1). Within 48 hours massive edema developed with proximal hyperemia. Gangrene developed extensively (Fig. 2). Eventually, he lost most of both feet and both hands.

The Gradients of Severity of Cold Injury

In frostbite from whatever cause, the injured member becomes white. Upon thawing, the sequence termed "the triple response" by Sir Thomas Lewis[23] is observed. This consists of local skin reddening followed by a

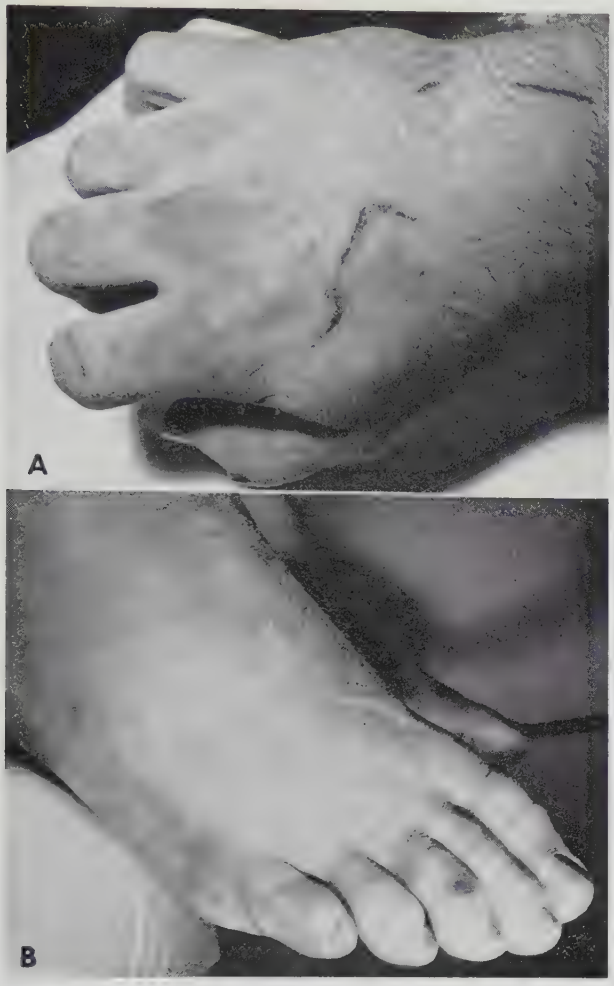

Fig. 1 (Case I). Hand and foot 24 hours after frostbite.

wheal at the site of injury, and subsequently by a flare in marginal tissues. In severe exposure, injury to muscles, large vessels, tendons, periosteum, bone and nerves is common. The presence and extent of gangrene is variable.

Since the Korean War, it has been customary to classify frostbite according to the extent of tissue damage.[28]

1st degree—Erythema and swelling with no blister formation
2nd degree—Blister or bleb formation
3rd degree—Full thickness injury with gangrene but without loss of a part
4th degree—Complete necrosis with loss of a part

Fig. 2 (Case I). Two weeks after injury.

Such a classification, which must be made in retrospect, is of limited value in assessing prognosis or planning care of the acute injury.

ETIOPATHOGENESIS

Individual Susceptibility as a Factor

Historically, certain groups of people have been noted to have an increased susceptibility to frostbite. This was recorded by Hippocrates[1] in his tract, "On Airs, Waters and Places," and in detail by Baron Larrey in his memoirs of the Napoleonic campaigns.[21] Modern epidemiologic surveys have substantiated the role of psychic and personality factors in frostbite. Neurasthenic, poorly adjusted, badly motivated or excessively sweating soldiers were more prone to cold injury.[37] Such factors were especially prominent in the 20 cases treated at the Denver Veterans Administration Hospital. Eleven patients had histories of chronic alcoholism and were inebriated when frozen. Of the combined series of 54 from both hospitals, 21 were intoxicated at the time of exposure. Generally, the patient went to sleep in the snow, in a car, or in an abandoned or unheated garage or house.

Case II (Denver VAH, A 5478). A 36 year old man was admitted 2 days after having become intoxicated and sleeping for 12 hours in an abandoned car. Outside temperature was −2° F. Army discharge diagnoses included anxiety neurosis and conversion hysteria. He was a known alcoholic. Gangrene of the tips of all toes developed. Eight months hospitalization was required for debridement and eventual multiple toe amputation.

Eleven of the 54 patients had been previously diagnosed as psychotic, most being chronic alcoholics in addition. The psychiatric diagnoses were

either schizophrenia or paranoia. A recurrent theme, both in patients with and without a frank psychiatric disorder, was an unconcern about self-protection, as exemplified in Case III.

Case III (Denver VAH, A 3808). A 46 year old man was admitted on December 6, 1960, for the treatment of frostbite incurred while walking barefoot in the snow 5 days previously. Most of his toes were absent as the result of two prior frostbite incidents in 1958 and 1959. These had resulted from sleeping outdoors and walking in the snow without shoes. He was a known alcoholic with a duodenal ulcer. A previous psychiatric opinion was that he "indulged in willful self-destruction." After a few hours, he released himself from the hospital. Five days later, he was found dead in the outskirts of a nearby town.

It should not be concluded from the foregoing remarks that all the patients were badly adjusted or inclined to place themselves in danger. In many cases, the exposure was obviously quite unavoidable. One 27 year old man was trapped beneath his car for 13 hours after an accident on a lonely road. Two nurses were frostbitten when forced to walk to safety from stalled cars. Varying degrees of frostbite are common among dedicated outdoor sports enthusiasts.

The importance of racial susceptibility has yet to be clarified. Studies in the Korean War indicate an increased risk of cold injury in Negroes.[28, 36] Conversely, Eskimos, Arctic fishermen and other inhabitants of northern geographic areas apparently have greater resistance to cold despite the fact that there is no consistently objective measurable difference in their response to reduced temperatures.[7, 14, 15, 26] It has been suggested that there is a tissue adaptation at the cellular level in response to the cold environment. Clever experimental proof of the value of cold acclimatization in the prevention of serious frostbite has recently been presented by Shikata, Shumacker and Nash.[34] Rabbits kept at 21° F. for 50 days were found to be much less seriously injured by a standard frostbite than control animals which had been maintained in the usual laboratory domicile. Studies on human acclimatization to cold have been inconclusive.[26]

Environmental Factors

The final determinants of the severity of frostbite, excluding individual sensitivity as mentioned above, are the degree of cold penetrating the tissues and the length of time this cold is applied to the affected part.[24] The environmental factors determining final tissue temperature are well known (Fig. 7). The first is the ambient temperature. In rabbits it was found that frostbite occurred where deep digital thermocouples indicated a temperature of less than 22° F.[20] This figure is consonant with a study of Korean War injuries in which about 90 per cent of the cold injury cases occurred below 22° F.[6] Of collateral importance are factors that increase

Fig. 3 (Case IV). Eight days after cold exposure.

heat loss by either conduction or convection, i.e., wetness, wind[27] or contact with metal.

Case IV (Denver VAH, A 616). A 52 year old man entered the hospital 6 days after cold exposure of his hands. Without gloves, he had assisted in pushing a stalled car during subzero temperature. Superficial skin slough resulted, not requiring special treatment (Fig. 3).

Extensive studies by the armed forces have shown that the most effective clothing consists of layers of wool cloth beneath an outer garment of tightly woven windproof fabric. This makes use of the insulating properties of still air.[35] In an analysis of various patients, neglect to

Fig. 4 (Case VI). Seven days after frostbite.

cover some discrete area is almost always the chief factor in determining the pattern of injury.

Case V (Denver VAH, A 12676). A 49 year old white man, while under the influence of alcohol, slept in −10° F. weather for several hours in his unheated car. His left shoe and sock were off during this time. He subsequently suffered severe frostbite with gangrene of several toes on the exposed extremity, with no apparent injury to his covered foot. His wife, who occupied the car with him and was likewise inebriated, was adequately protected by clothing; she suffered no injury whatsoever.

Case VI (CGH, Outpatient). A 10 year old Negro boy was seen in the emergency room 5 days after playing outdoors in subzero temperature while wearing poorly fitting trousers. There were rings of frostbite on the legs at the exposed area between his socks and the bottom of the trousers (Fig. 4). Therapy was not required.

Mechanism of the Injury

The consequence of frostbite is cell injury or death. The mechanism of this injury has been the subject of important controversy, since it bears on the type and potential efficacy of therapy. If, as Lewis,[22] Meryman[25] and many others believe, frostbite is essentially a thermal injury, the principal tissue damage has occurred by the time of rewarm-

ing and any or all subsequent measures will do little to alter the progression of pathologic events. If, however, vascular or other factors are of fundamental significance in the production of cell damage, there might be more hope for effective treatment. A brief description of the observed phenomena in experimental frostbite may be of interest in evaluating these unsolved questions.

Studies in cooled extremities suggest that a profound reduction in blood flow occurs promptly with cold exposure. When the arms of human volunteers were immersed in water at 55° F. for two hours, blood flow was reduced to approximately 3 per cent of control levels.[3] At 32° to 46° F. there is virtually no circulation in the fingers.[17] Although cooled tissues require less oxygen, it is reasonable to assume that the reduction in flow is of such extent as to result in ischemia[25] and eventual tissue death. Such a mechanism could explain gangrene in nonfreezing cold injury, and it would be an important stage in the development of frostbite at colder temperatures.

Many authors have observed the changes after experimental frostbite, but the well-controlled studies of Kulka[19, 20] and Bellman[4, 5] provide the basis for much of the following description. During actual freezing, the tissue becomes opaque as observed by transillumination.[11] Upon thawing, appearance is normal for a short time. Edema and rupture of small vessels is then observed, particularly of small venules.[5] The role of the protein-rich products of extravasation and transudation are unknown, but it has been suggested that they cause immediate vascular compression and ultimate fibrosis.

Changes in small blood vessels are obvious promptly with thawing. Using microangiographic and transillumination techniques, these may evolve for weeks or months.[5] There is an immediate, intense arterial vasoconstriction at the junction of uninvolved tissue with frozen tissue. Within a few hours, a demonstrable increase in vascularity occurs in the marginal zone, at the time this area becomes clinically erythematous. Some authors have described the opening of small arteriovenous shunts in this region.[20] Flow rate is reduced and intravascular sludging is observed. Within three days multiple small areas of stenosis, or frank, sharply delineated occlusions have been observed microangiographically[5] within the previously frozen area. Tubular filling defects which are thought to be thrombi can be demonstrated in both arteries and veins. By the tenth day these either regress or proceed to stenosis or occlusion of the vessels. During the ensuing weeks, both arteries and veins disappear from the microangiographic pattern, presumably due to late thrombosis. The resulting destruction of vascular architecture is partially compensated by the previously mentioned increase in marginal blood supply from which new branches may bud into the formerly frozen area.[5] However, in six to twelve weeks such new networks undergo involution

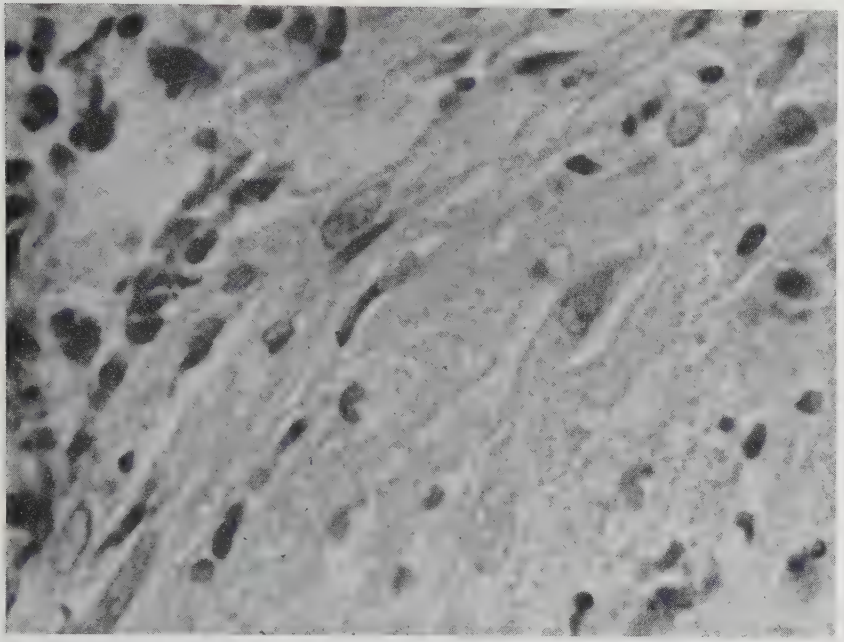

Fig. 5. Arterial injury, 1 month after frostbite. Lumen is at upper left. Note intramural edema, disruption of epithelieal lining, and thrombus.

and, if the injured area does not undergo necrosis, it is left with a distorted and cross-sectionally reduced vascular pattern.[5] Histologic sections at various stages in the above sequence show edema and small-vessel thrombi which are often fibrinoid in character (Fig. 5), and later areas of local arteritis or intimal proliferative thickening (Fig. 6).

The ultimate result of various types of cold injury, as evaluated by histologic study, is essentially identical. This does not, of course, prove that all varieties have an identical pathogenesis. Frostbite differs from other cold injuries in that there is the possibility of actual tissue freezing. How often this occurs clinically is difficult to determine, since virtually all patients have been warmed before they are seen by a physician. Some experts believe freezing is uncommon. Others, particularly Meryman,[25] believe that this is a common event in frostbite, is a factor of major significance, and justifies the sequestration of frostbite as a separate variety of cold trauma.

It was formerly believed that ice crystal formation within the cells led to their death by simple mechanical rupture of the cell wall. Meryman[25] observed that ice crystals develop exclusively from available water in the extracellular compartment rather than within the cell. Under these conditions, a hypertonic environment gradually forms about the cells. Intracellular fluid then enters the interstitial spaces producing,

Fig. 6. Arterial injury, 11 months following 2° frostbite. Note fibrous intimal proliferation and perivascular lymphocytic infiltration (Case IX). Lumen is on the right.

in effect, a local type of intracellular dehydration with ultimate cellular death.

Figure 7 summarizes the probable sequence of events in the pathophysiology of frostbite. As discussed above, there is some experimental evidence that each of these pathways contributes to the degree of injury. Their relative importance is not as yet ascertained, and more research remains to be done in this area.

TREATMENT OF FROSTBITE

Lack of understanding of the pathophysiology of frostbite has made it difficult to recommend or follow rigid principles of therapy. In general, treatment can be divided into measures related to the local care of damaged tissue, and measures designed to diminish secondary vascular damage.

Method of Rewarming the Frozen Part

Until a few years ago, it was thought that rewarming of the frostbitten member should be done slowly. Rubbing the feet with snow, walking, beating the injured part, and plunging the extremity into

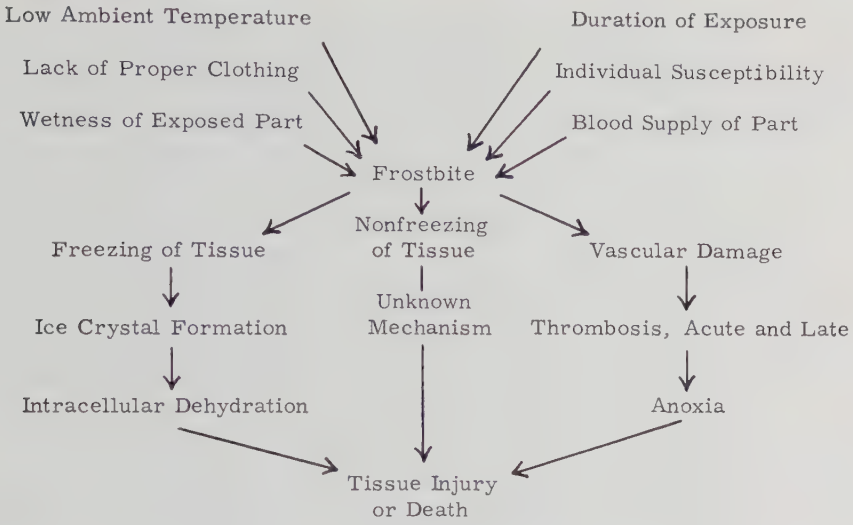

Fig. 7. Pathophysiology of frostbite.

cold water baths were variants of this approach which were not only passed along from generation to generation among the lay population but actively taught to first aid groups by physicians. A more temperate approach is to allow the patient to warm slowly at room temperature.

Studies with experimental frostbite suggest that the greatest salvage is with rapid warming of the affected part. Decisive information on this point in experimental frostbite has been provided by Lewis[22] following earlier work by numerous investigators.[2, 10, 11, 13, 31] Unquestionably, the major benefit of rapid warming is due to reduction in total time of cold exposure. In addition, there is some suggestive evidence that rapid rewarming has inherent value quite apart from mere decrease in freezing time.[16]

The conditions and details of rapid rewarming are explicit.[22] The optimum temperature is 104° to 107.5° F. (40° to 42° C). The frozen extremity is placed in a water bath at this temperature. If the water bath is heated to more than 107.5°, harmful rather than beneficial effects occur. If the temperature is less, even by a few degrees, maximum benefit is not obtained. Heavy sedation may be required during the actual rewarming.

Since most patients with frostbite have already been warmed before seeking medical treatment, the greatest advance in immediate care can be made only in the education of the public and in lay groups trained for first aid duty. It is also necessary within hospitals to adopt a consistent policy applicable to those unusual patients exemplified by Case I, in which instance rewarming could have been performed under the ideal circumstances of a hospital environment.

The Value of Sympathectomy

A consideration of the pathophysiology of frostbite does not provide much hope that sympathectomy will profoundly alter the progression of changes. The acute vasoconstriction that occurs at the time of cooling regresses promptly with thawing, and is replaced with vasomotor dilatation and hyperemia of the marginal zone. Denervation would appear to offer little augmentation of blood supply. Controlled experimental studies provide equivocal or no evidence that sympathectomy is of value. There have been some clinical reports of benefit from sympathectomy. The most extensive clinical study was by De Jong and his associates,[9] who described controlled evaluation of sympathectomy in 19 patients with bilateral frostbite of either the hands or feet. These authors performed unilateral sympathectomy and compared the subseqeunt behavior of the treated versus the nontreated limb. They observed more rapid resolution of edema and demarcation of slough of the denervated extremity, providing the sympathectomy was done within ten days of the time of injury.

In two patients in the present series with symmetrical injuries of the feet, sympathectomy was performed on one side. There was no difference in outcome of the treated feet compared with the nontreated feet.

Case VII (Denver VAH, A 12676). A 27 year old schizophrenic male was admitted 12 hours after having walked in the snow without shoes for 4 hours. During the ensuing 6 days, it was clear that the involvement of the feet was absolutely symmetrical. After 6 days, a left lumbar sympathectomy was performed. The development and evolution of gangrene subsequently was exactly the same in the two feet (Fig. 8). The course was not influenced in any demonstrable way. Initially, it was feared that extensive tissue loss was inevitable. After two and a half weeks, a black eschar sloughed bilaterally, revealing pink epithelium.

The Value of Intra-arterial Vasodilators and Sympathetic Block

Numerous reports have stressed both the value and the lack of effect of these therapeutic approaches. The conceptual weakness of these forms of therapy would seem to be similar to those of sympathectomy. A specific effect of intra-arterial sympatholytic drugs such as Priscoline in relief of pain has been noted, and will be mentioned again under the section on pain.

The Place of Anticoagulants, Fibrinolysins and Low Molecular Weight Plasma Expanders in Therapy

As mentioned previously, prominent features of the frostbite injury are progression of vascular changes long after the acute injury involving sludging and the formation of agglutinative thrombi. The use of the

Fig. 8 (Case VII). Eighteen days after frostbite.

above agents in the therapy of acute frostbite would appear logical. Heparin has received the greatest trial, with equivocal results, both in this series and elsewhere.[18] Low molecular weight plasma expander, which has been shown in other circumstances to prevent blood sludging,[12] and fibrinolysin, which might reverse the thrombotic process,[33] have not been evaluated.

Local Care

Frostbite injuries have been treated with dressings and by the exposure method. The results appear to be little different, provided the techniques are meticulously executed. In this series, the open technique was used in most cases, with bacterial isolation of the patients. Dry

gangrene and slough developed in almost all cases, without the complication of secondary infection.

Exercise of the Afflicted Extremity

During the acute phase of injury, it seems reasonable to immobilize the injured part. As soon as acute inflammation has subsided, exercise should be instituted. This is mentioned in Simeone's analysis of war cold injuries[37] as one of the few measures of undoubted therapeutic efficacy. In the present series it was not unusual to see good motion, and eventually good function, of blackened fingers and toes. Immobilization will result in stiffening and fibrosis in a member which could otherwise achieve functional restoration.

Indications for Amputation

One of the characteristic features of frostbite is the surprising salvage that is possible in an apparently badly injured extremity. Final classification of the extent of injury is possible only in retrospect. A blackened and apparently hopelessly traumatized foot may turn out to be almost completely preserved with only superficial skin slough. Extirpation should, therefore, be done with great conservatism and as late as possible. As long as there is movement in the involved area, there is hope for preservation of tissue. In the present series of 54 patients, there were 60 amputations. However, most of these were toes and fingers, almost always preserving more tissue than was expected early in the course. There were also two forearm amputations, two hand amputations, six foot amputations, seven below-knee amputations and two above-knee amputations. The above-knee amputations were in patients with associated occlusive vascular disease.

Pain Control

The control of pain in most patients of this series has not been a serious problem. Generally, aspirin or codeine alone is sufficient. In numerous others, paraldehyde, chloral hydrate and promazine were used, but alcoholism was the underlying indication for these drugs. In an occasional patient with early severe causalgic pain, one of us[29] has used intra-arterial Priscoline with apparent relief. The mechanism of this relief is not clear. A similar effect has been noted by Shumacker[35] following sympathectomy. It should be re-emphasized, however, that most patients do not require unusual measures for control of pain.

Long-Term Sequelae

Other than obvious loss of portions of limbs or areas of skin from gangrene, frostbite can precipitate a long-term illness with a disabling array of physical findings and symptoms. The following example typifies an all-too-common sequence of events following severe exposure to cold:

Case VIII (CGH, 423145). A 37 year old white man suffered third degree frostbite of both feet and hands 3 years prior to admission. He sought no medical care at the time. He had had progressive circulatory embarrassment since then, with numbness and coldness in all extremities, most marked in winter. Two years following injury, he lost the tips of two toes and the right index finger because of vascular insufficiency. On presentation here, he had impending gangrene of the tip of the right middle finger, his hands and feet were cool and moist, and the skin was shiny. The right third finger and two toes on the left foot showed cyanotic rubor. He underwent a right cervical sympathectomy and bilateral lumbar sympathectomy with good relief of symptoms.

Causalgic-type pain, hyperhydrosis, coldness of extremities and stiff joints were among the most common sequelae noted by Blair[6] in a four-year follow-up of Korean War casualties. In general, the degree of long-term disability was related to the severity of the original injury, and discomfort was greater in cold weather than in warm weather. These patients are also much more susceptible to a second cold injury, having once been exposed.

Case IX (CGH, 37387). A 57 year old white man sustained a minor exposure to cold 11 months following a second-degree frostbite of both feet. He suffered increasing causalgic-type symptoms in both feet following the second exposure. There was dependent cyanotic rubor and hyperhydrosis. These symptoms were relieved by lumbar sympathectomy. In addition, the distal phalanx of his fourth toe was amputated because of vascular insufficiency. The arteries in the toe were seen to contain fibrous internal proliferation, with perivascular lymphocytic cuffing as evidence of a long-standing, low-grade vasculitis (Fig. 6).

That these long-term alterations are secondary to the vascular injury sustained at the time of exposure has been well accepted. In addition, physiologic studies by Simeone have shown diminution of the ability of these vessels to react to stress. Blood vessels studied in the skin did not constrict as effectively as the normal ones did when exposed to cold, and did not dilate as effectively when vasoconstriction was blocked.[37]

Another aspect of long-term frostbite injury was recently reviewed by Ritchie.[30] In a study of 13 individuals examined four to 50 years after being frostbitten during childhood, it was noted that frostbite had damaged the epiphyses and resulted in lack of development, deformity and limited use of the fingers. The loss of function was in general related to degree of exposure and age at the time of the episode.

PROPHYLAXIS

Rarely does the well worn maxim, "an ounce of prevention is worth a pound of cure," have more immediacy and meaning than in the prophylaxis of frostbite. Once the cold injury has taken place, no treatment

can save cells already destroyed, and subsequent therapy is essentially supportive.

The realization that frostbite implies a long-term illness with possible economically disastrous consequences should be impressed on all people who live in cold regions. This is most important in outdoor recreational activities. Frostbite is a silent assailant. This fact should be thoroughly appreciated by the public. Lack of pain at the time of injury is part of the natural history of the disease.

A pitfall for the unwary, in Colorado at least, is the lack of feeling of cold despite low ambient temperatures. Rocky Mountain folklore relates this to low humidity, but, as Sir Thomas Lewis has pointed out,[24] the humidity of air with freezing temperatures is essentially zero irrespective of geographic location.

Proper clothing, which means multiple layers of light woolen material and a windproof outer garment, are basic equipment. Proper head gear and foot wear are extremely important. Gloves should be worn at all times, particularly when the hands must come in contact with metallic objects.

Unfortunately, a sound educational and public health program will have little meaning for the majority of our patients. Their sociopathic and often frankly psychotic backgrounds preclude any worthwhile value in this area.

SUMMARY

Frostbite is a serious illness with potentially disastrous consequences which are often not apparent immediately after injury. Factors contributing to the severity of the injury have been enumerated.

The pathologic physiology of the injured member following exposure has been intensively studied in laboratory animals with subsequent extrapolation to human cold injury. That much still remains to be understood is evident by the diverse modes of treatment, each with its proponents. Certain aspects of therapy have, however, become generally accepted. If thawing of the frostbitten extremities has not taken place at the time the patient is first seen, the affected parts should be warmed rapidly at a temperature of 104° to 107.5° F. (40° to 42° C). Open treatment is as effective as dressings. Ambulation should be started as early as possible.

In the absence of infection, amputation should be delayed until definitive demarcation has occurred. This may and usually does take several months.

In a civilian population living in cold climates, a public health education program would be of some benefit. However, because of the

emotionally disturbed condition of the patient in almost half of our cases, the value of this form of prophylaxis is limited.

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Bites—Human, Dog, Spider and Snake

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In the thirteenth century the eminent philosopher-surgeon Arnold of Villanova started his treatise on poisons with this sentence: "In this book I propose, with God's help, to consider diseases of women, since women are poisonous creatures. I shall then treat of the bites of venomous beasts." Since those days the specialties of gynecology and toxicology have been more clearly defined and this paper will discuss only the second of Arnold of Villanova's topics, except, as will be seen below, to describe how the bite of a woman may be as severe as that of a venomous beast.

The bites of animals and insects are among the commonest types of trauma to which man is subject. The number of insect bites each year is uncountable—about 500,000 persons each year in the U.S.A. are bitten by dogs and from 2000 to 3000 each year by snakes. The bites of horses, cattle, cats, bats, foxes, skunks, squirrels, sharks, barracuda and humans are all recorded regularly and can give rise to every degree of morbidity from the trivial to the catastrophic. Not only may the bites themselves injure and deface, but the injection of poisons may be more dangerous than the bite itself and superimposed infections may seriously increase the resultant damage and prolong the period of disability. In addition, the public health and socio-economic aspects of bites are tremendous. About 40,000 to 50,000 persons each year in this country receive prophylactic anti-rabies injections, each patient receiving an average of 14 injections. Thousands of dogs and cattle are destroyed every year because of suspected rabies, and the toll of psychological trauma to victims and their families is beyond assessment.

Several types of injury will be discussed and salient points in their management illustrated by case histories.

HUMAN BITES

Human bites can be regarded under three headings. First, a genuine bite in which the assailant sinks his teeth into his victim producing a puncture wound or laceration and, in many cases, causing actual loss of tissue—the tip of a nose or an ear—to the victim. Second, the injury

inflicted, usually to the knuckles, on an assailant who strikes his victim on the mouth. This type of injury, while perhaps being more socially and esthetically acceptable than the first type, can nevertheless result in severe injury. Third, in which a person bites himself. This injury is almost always on the tongue or lip and usually occurs when the patient falls and strikes his chin. It is not uncommonly sustained by an epileptic during a grand mal seizure.

The human mouth, in spite of the best of modern oral hygiene, seethes with a variety of pathogenic bacteria—staphylococci, streptococci, Vincent's bacilli, spirochetes and others.[4] In contrast, the mouth of a healthy dog is remarkably free of these pathogens and the expression "as clean as a hound's tooth" is not without validity. Because of the numerous pathogens harbored in the mouths of humans, the main complication of human bites is secondary infection. Heavy contamination of the wound should be assumed and treatment directed toward its eradication.

The examination of a bite wound must include an evaluation of the damage done to deep structures.[1, 2] If the wound is on the knuckles, injury to the extensor tendons may be concealed by retraction of the severed tendon. Therefore, the patient should be asked to "make a fist" and the wound examined in that position. The wound should first be cultured, then thoroughly cleansed by a hexachlorophene soap scrub for ten minutes, and finally liberally irrigated with sterile saline.

Damaged tissue should be debrided according to accepted surgical principles. Severed tendons and nerves should not be primarily sutured, but if the wound involves only soft tissues, is seen early, and is adequately cleansed and debrided, primary closure may be permissible. Large doses of antibiotic should be administered systemically. The selected antibiotic may have to be changed after therapy has started if any organisms grown from the initial culture are not sensitive to it.

If the hand is injured it should be maintained in the position of function, using a large bulky dressing, until all signs of infection have subsided. Severed tendons, if present, should then be repaired in the usual manner.

Bites of the face often involve the lips, nose or ears, and a large percentage require plastic reconstruction because cartilage is lost and a deformity is present. This type of reconstructive surgery has recently been described by Curtin and Greeley[3] and, in all cases of this sort, the advice of a plastic surgeon should be obtained. Attempts to reattach totally avulsed portions of the nose or ear are likely to end in failure.

CASE I. This young Spanish-American male, aged 24, was in bed with his girl friend when a quarrel started. In the ensuing altercation his lady-love bit him on the medial aspect of the right thigh, midway between the hip and knee. When he first reported to the hospital 4 days after this incident, the wound was swollen

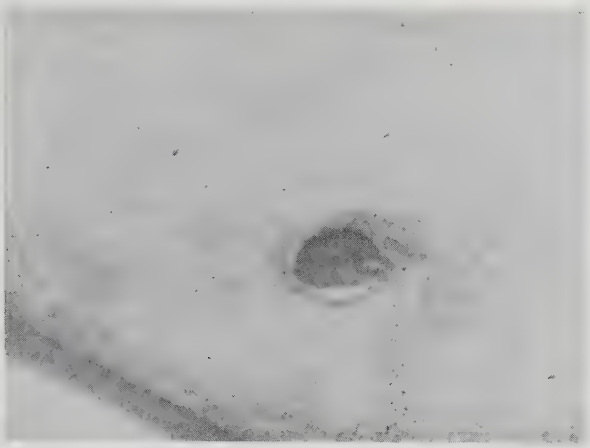

Fig. 1 (Case I). Granulating, slowly healing ulcer of thigh caused by a human bite.

and obviously infected and the regional nodes were enlarged and tender. In the following few days the wound, which had initially presented as a laceration, turned into a circular ulcer and subsequently healed slowly by granulation (Fig. 1).

Accidental self-inflicted bites of the tongue may be quite serious. The wound is usually gaping and may bleed considerably. These wounds are often difficult to clean and because of their situation are constantly bathed in a stream of infected saliva. After cleansing and debridement they should be sutured lightly. Sutures are especially necessary if the edge of the tongue is involved. It is advisable to cut the ends of the sutures long, because the constant movement of the tongue may undo a knot which is cut off too short. Cleansing mouthwashes should be used frequently until the wound has healed.

An unusual complication of a wound of this sort is illustrated by the following history.

Case II. This small Spanish-American boy, aged 2, was first admitted in January, 1957, with a swollen infected bite of the tongue which had been sustained 2 days previously. No attempt was made to suture the wound because of the infection, and with systemic antibiotic therapy the wound healed. However, in the next 18 months, this child was readmitted on 5 separate occasions with intermittent swelling of the tongue in the area around the scar of the first trauma. On the third of these admissions, the area was biopsied and a submandibular lymph node was also excised for examination. Histological appearances of scar tissue and chronic infection were noted. Subsequently, the tongue became severely swollen again and eventually the child underwent an excision of the anterior one-third of the left side of the tongue. With complete examination of this specimen, the diagnosis of cavernous lymphangioma was made. In view of the development of this lymphangioma in the precise area where the original injury was received, it is difficult to dissociate the two events.

DOG BITES[5, 7, 9]

About half a million people are bitten by dogs each year in the U.S.A. In Denver 1757 persons were bitten by dogs in 1960 and 1912 in 1961. Most persons bitten by dogs are children, only about 2 per cent being the traditional mailmen. Many of these bites are received when trying to separate fighting dogs, when teasing or annoying dogs, or by children who try to take away a dog's food while he is eating. Genuinely unprovoked attacks are relatively rare and should be viewed with suspicion, because such is the behavior of the rabid animal. Much could be done to prevent dog bites if parents taught their children how to handle and respect not only their own but also other people's pets. It is as stupid for parents to teach their children that all dogs are "nice little doggies" to be immediately petted and fawned upon as it is to teach them that all dogs are to be feared. Many a child has been bitten when overtaken by a playful dog from which it is trying to run away in misplaced fear. Small puppies bite more frequently than older dogs and the combination of a small untrained puppy and a young, rough, teasing and thoughtless child will inevitably end in the child being bitten.

The immediate management of dog bites follows the principles of treatment of any laceration. The extent of the injury must be assessed by examination. The wound is then thoroughly cleansed with soap and water and irrigated. Debridement may be carried out if necessary. Because dog bites are relatively "clean," the wound may often be closed primarily after cleansing and debridement. Anti-tetanus therapy should be given in addition to antibiotics.

Rabies

The main fear following dog bites is infection with rabies. Approximately 40,000 to 50,000 people receive anti-rabies prophylaxis each year and 10 to 20 persons die each year in the U.S.A. from rabies. The dog is the commonest source of human rabies but is not as frequently infected as some wild animals such as the fox. All warm-blooded animals can be infected with rabies, and the view that cat bites (Fig. 2) do not give rise to this disease is erroneous. Domesticated cats, however, are not commonly in contact with other rabies-carrying animals. Skunks, bats, squirrels and raccoons all commonly carry rabies. While it is impossible to eradicate rabies completely from both domesticated and wild animals in any community, a great deal can be done to reduce the incidence to minimal proportions. For several years Denver, Colorado, has had a dog vaccination regulation, and probably more than 70 per cent of the dogs in this community have been vaccinated against rabies. As a result, there has not been a case of dog or cat rabies in Denver since 1950, and there has never been a case of human rabies at Colorado General Hospital.

Fig. 2. Arrow points to puncture wounds caused by an unprovoked cat bite. Breakage of the skin constituted genuine exposure.

In order to develop rabies a person must receive a direct inoculation of the virus subcutaneously. Usually this occurs as a result of the bite of a rabid animal, the saliva of the animal being heavily infested with the virus. However, under unusual circumstances a patient may be infected in other ways.

Case III. This 60 year old man, a biologist at a veterinary college, was seen in January, 1960. He had been dissecting the cadaver of a mouse known to be rabid when he accidentally pricked his finger with the blade of the scalpel. He came immediately to the hospital. The local lesion required no treatment, but he was given a single dose of hyperimmune serum and was started on a 14-day course of duck embryo vaccine. The course of prophylactic injections was completed without complications. There is no evidence that actual infection occurred, but the potentialities of the injury require no emphasis.

Local Treatment of the Bite. The local treatment of a bite thought to have been inflicted by a rabid animal has been the subject of some controversy. The traditional treatment has been to cauterize the wound with fuming nitric acid. The efficacy of this somewhat barbaric treatment is doubtful. There is some evidence that rabies virus can very rapidly become attached to neural tissue, although its generalized dissemination takes somewhat longer. Moreover, fuming nitric acid is not a chemical to be found in the average doctor's office. If the wound is of a deep puncture type accessible only to investigation with a probe, there may be a case for the use of nitric acid. If, however, it is extensive and amenable to direct cleansing and debridement, or if it is on the face, acid should not be used because it results in further necrosis and the development of extensive scarring. Bleeding of the wound should be

encouraged and in a very suspect case hyperimmune serum may be injected around the wound.

INDICATIONS FOR ANTI-RABIES TREATMENT. When should a patient be given anti-rabies treatment? This is the question facing any doctor called to treat a dog bite. The World Health Organization Committee on rabies has published an outline of the course of action to be followed in any case of possible exposure to the disease.[6, 8] The exact details of these criteria may be found elsewhere. However, as a general guide, it may be stated that rabies prophylaxis should be administered under the following circumstances:

(1) If the dog is shown by pathological examination to have rabies.
(2) If a clinical diagnosis of rabies without histological confirmation is made by a veterinarian.
(3) If an unprovoked attack is made by a dog and the dog escapes or is killed and the body destroyed without the opportunity for an examination.

In any of these circumstances, rabies prophylaxis should be started without delay.

RABIES PROPHYLAXIS. If the attack is known to have been made by a rabid animal, hyperimmune serum is injected around the wound site after suitable tests of sensitivity of the patient to the serum have been made. At the same time, the patient should be started on a 14 to 21 day course of vaccine. A number of vaccines have been available but it is now generally recognized that duck embryo vaccine is less likely to give rise to serious complications than the older forms of vaccine. If an animal is shown to be rabid some days after the bite has been inflicted there is probably not much point in giving hyperimmune serum at that time and the course of anti-rabies vaccine should be started without it. The anaphylactic and allergic responses to hyperimmune serum are not insignificant and it should not be used haphazardly when the benefit to be gained from its use is negligible. Children under five years of age are more susceptible to rabies than older persons and for them the indications for starting anti-rabies vaccination are less stringent than for adults. Licking of the intact skin by a dog does not constitute exposure. However, if a dog thought to be rabid licks freshly abraded skin this constitutes dangerous exposure. If it licks crusted abrasions the danger is problematical and the judgment of the doctor must prevail.

If the animal remains healthy after seven days of treatment to the patient there is probably no need to continue the course of treatment for the full 14 days. However, if the animal becomes ill between the fifth and the tenth day of treatment to the patient, then the patient should probably receive a second dose of antiserum and should have the course of treatment extended to 21 days. If the animal is still healthy

Fig. 3. Negri body within a cerebellar cell. Specimen from a dog's brain.

after ten days of treatment to the patient the possibility of rabies can be almost totally excluded.

Diagnosis of Rabies in the Animal. The precise diagnosis of rabies in the animal can be made from a history of exposure of the animal to other animals with rabies, by the clinical signs and symptoms, by the course and termination of its illness, and specifically by the demonstration of Negri bodies in the cytoplasm of the nerve cells (Fig. 3). Inoculation of cerebral tissue into other animals can be done to further confirm the diagnosis, but this involves a three to four week delay. If an animal suspected of having rabies dies and immediate facilities are not available for an autopsy and examination, the head of the animal should be removed and either taken to the nearest competent laboratory or refrigerated until arrangements are made for pathologic examination of the brain.

Course of Rabies in the Human and in Dogs. In humans, rabies is invariably fatal. The course of the disease is rapid. For two to four days there are prodomal symptoms before the phase of excitation is reached. The patient is then nervous and unable to sleep and has hyperesthesia and frequent spasm of the throat muscles. Drooling and maniacal behavior supervene until the final stage of coma and paralysis ends in death.

In dogs, there may be very few pathognomonic signs. Classically the disease has been described as either "furious" or "dumb." Animals with the furious type of rabies are most likely to transmit the disease, and

during their illness they have purposeless roaming activity, snapping in an unprovoked manner, drooling, and vocal cord paralysis. Death usually occurs within two to five days.

SNAKE BITE[10, 11]

Many areas of this country contain large numbers of snakes, and with the increasing popularity of camping, fishing, hunting and other outdoor sports it is not surprising that several thousand people are bitten by snakes every year.[13] In the minds of most people, the bite of a snake means a high likelihood of death, but actually the mortality from snake bite is low, with only 10 to 20 deaths per year in the U.S.A. from snake bite.[12] Nevertheless, there is probably a more generalized and inbred fear of snakes than of any other animal.

Sensible awareness of the possibility of being bitten by snakes would certainly decrease the incidence of this type of accident. This is illustrated by the following two case histories.

CASE IV. This 12 year old boy was out hunting for lizards in an area where snakes were known to be frequent. A lizard he was chasing ran into a bush and, instead of trying to extract the lizard with a stick, he put his hand under the bush and was promptly bitten by a snake.

This boy was old enough to understand the danger of snake bite and should have been taught not to put his hands into holes or under rocks where an unseen snake might be lurking.

CASE V. This small boy, aged 5, lived in the same area as the patient described above. His parents stated that the child frequently went barefoot into the fields. One day, while out running, he trod on a small rattlesnake and was bitten. He was admitted to the hospital and had a fairly serious local reaction, but fortunately was able to leave the hospital after two weeks without any serious complications.

This child was perhaps too small to realize the danger in what he was doing but his parents should have insisted that he wear shoes and trousers.

The possibility of death or serious complications from snake bite depends upon such factors as the age and size of the patient, the size of the snake, the site of the bite, the immediate treatment instituted, and the definitive treatment given when medical help is available. Most bites occur at places some distance from a doctor or hospital, and it is common for several hours to elapse between the time of the bite and the first treatment by a doctor. There is a fairly widespread realization among the public that the immediate form of first aid is a tourniquet placed proximal to the bite. However, there is little knowledge of the correct tightness of the tourniquet, and there have been numerous

Fig. 4. Contents of standard commercially available snake bite kit.

instances of severe ischemia to the limb resulting from the prolonged application of too tight a tourniquet. It has also been widely accepted that the site of the bite should be incised. The most commonly available snake bite kit contains a rubber tourniquet, a suction device, a razor blade and instructions in the use of all three* (Fig. 4).

In recent years, there has been some controversy about the correct first-aid treatment for snake bite. The most widely accepted measures have been tourniquet, local incision, the application of suction, proximal circumferential incision, the application of ice, the rubbing of potassium permanganate crystals into the wound, and the consumption of stimulants, usually whiskey. There have been vociferous protagonists and antagonists for each of these methods of treatment. The arguments have been based not only upon clinical but also upon experimental evidence, and the inescapable fact is that the majority of people in this country bitten by snakes would probably recover no matter what treatment is given.

The general basis for the use of these various types of treatment is that the snake injects into the subcutaneous tissues a variable quantity of venom, which is then absorbed by the lymphatics. By the application of a tourniquet this absorption is delayed, and by the incision of the

* Made by Becton-Dickinson & Co., Rutherford, N. J., $2.25.

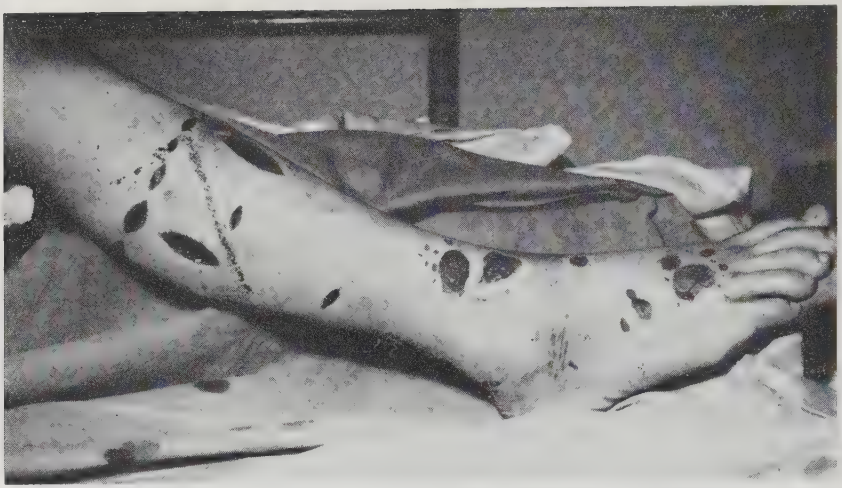

Fig. 5. This picture was taken 48 hours after fasciotomies had been made (vertical incisions). The incisions made at the time of initial treatment (circumferential) and the blebs on the foot are readily seen.

wound and the use of suction some of the venom may be sucked out before it is absorbed. However, if the incision is not made deeply enough no amount of suction will extract the venom. It has been said that the venom is usually injected as deep as three-fourths of the distance between the fang marks, and therefore, if an incision is to be made, it is probably best done by joining the fang marks by an incision equal in depth to the distance between the puncture wounds. Proximal incisions at the edge of the spreading area of inflammation and swelling have been advised, but the evidence that venom can be extracted by suction through these proximal incisions is very slight. Under some circumstances this type of treatment may, by itself, be almost as dangerous as the snake bite.

CASE VI. This boy, aged 9, was bitten on the right heel by a small rattlesnake. The bite occurred at 2 P.M. and the child's father immediately applied a tourniquet from a torn-up sheet, below the knee. The patient reached the emergency room of the hospital at 4:30 P.M., at which time there was some ecchymosis and visible swelling of the foot, and the fang marks were readily seen. The child was nauseated and vomiting. An incision was made locally between the fang marks and antivenin was injected both around the wound and intramuscularly. Later in the afternoon, numerous circumferential incisions were made below the knee by a resident who was a self-professed expert on the treatment of snake bite. By the next day, the leg distal to these incisions was grossly swollen and tense, and the skin was covered with numerous blebs. It was apparent that these numerous circumferential incisions had so impaired the lymphatic drainage from the limb that the edema in the limb was producing arterial insufficiency. A three-compartment fasciotomy was therefore carried out (Fig. 5). At this operation, the muscles bulged through the fascial incisions. Thereafter, there was considerable drainage of edema fluid from the fasciotomy wounds, and a fairly rapid re-

covery of the limb. The patient was discharged on the thirteenth postoperative day without further complications.

In this case, there was no question in the mind of the attending surgeon that the three-compartment fasciotomy was instrumental in saving the limb from serious damage. The following case history represents a situation in which fasciotomy should probably have been carried out.

Case VII. This 10 year old boy of Christian Science parents was pitching hay on his father's ranch when he was bitten on the left forearm by a rather large rattlesnake. A tourniquet was immediately applied around the upper arm by the boy's father. No incisions were made over the bite. The bite occurred at approximately 2 P.M. and the patient arrived at the emergency room of the hospital at 4 P.M. Antivenin was immediately given both around the wound and intramuscularly, and in addition penicillin, streptomycin and tetanus antitoxin. The site of the bite was incised and suction applied. The forearm was also cooled and because of profound shock an intravenous drip with Vasoxyl was started. Quite rapidly, the left arm and hand became about twice the normal size and were blue, tense and congested. The blueness deepened to a dark purple color and the swelling extended to the shoulder and supraclavicular region. The patient received a total of 6 ampules of antivenin and large doses of cortisone. His hematocrit dropped to 32 per cent, and after about 48 hours he complained of absence of sensation in the finger tips. This neurological deficit increased until there was a complete paralysis of the median and ulnar nerves and a flexion contracture of the fingers. In spite of this situation, the parents removed the child from the hospital after 10 days against medical advice.

If a fasciotomy had been performed on the forearm as soon as the first indications of neurological damage became evident, it is possible that the subsequent crippling contracture might have been avoided.

In all severe snake bites the area bitten becomes swollen and there may be a variable amount of local necrosis. Extensive swelling of the limb such as that described in the case histories above is not an uncommon response. Under these circumstances incisions should be made not in an attempt to release any toxins from the limb but purely as a means of decompressing the fascial compartments so that the pressure of the swollen edematous muscles will not give rise to permanent ischemic damage.

In spite of the controversy about the first-aid treatment of snake bite the following principles should probably be followed. A tourniquet of moderate tightness should be applied proximal to the area bitten. It is not necessary and, indeed, it is dangerous for this to be applied tight enough to occlude the arterial supply. It may be released for about half a minute every ten minutes. Blind cruciate incisions over the fang marks are probably inadvisable. They have been known to do damage and if they are self-inflicted the chances are that they are somewhat timidly made and shallow and therefore inadequate. Suction may be applied; it

ONE COMBINATION PACKAGE
PKG. 62A138
ANTIVENIN (CROTALIDAE) POLYVALENT
(equine origin)
(North and South American Antisnakebite Serum)
Wyeth
For neutralization of the venoms of North American rattlesnakes and moccasins—Crotalus, Agkistrodon and Sistrurus; and the venoms of the crotaline species of South America—as the fer-de-lance (Bothrops atrox), and the tropical rattlesnake (Crotalus terrificus).
ANTIVENIN (CROTALIDAE) POLYVALENT
ANTIVENENO OFIDICO (CROTALIDAE) POLIVALENTE
DISTILLED WATER
AGUA DESTILADA

Fig. 6. Contents of snake antivenin package. This package contains one vacuum vial to yield 10 cc. of serum—to be used immediately after reconstitution—(with preservative: phenol 0.25 per cent and sodium-ethyl-mercuri-thiosalicylate 1:20,000); one syringe containing 10 cc. of pyrogen-free distilled water (with preservative: phenylmercuric nitrate 1:100,000); one applicator vial of Iodine Solution, N.F.; one 1 cc. vial of Normal Horse Serum (diluted 1:10) as test and desensitizing material and one sterile, double-end needle.

may remove some of the venom and at least is relatively harmless. Ice may relieve some of the pain, but there is experimental evidence to indicate that it may also increase the necrosis.[15] It is true that in these days of "civilized" camping it is quite likely that ice may be readily available, but in many instances it will not be available. The limb should be kept as still as possible because immobility reduces the amount of lymphatic absorption. However, recovery may follow even if this is not possible.

CASE VIII. A sheepherder, aged 63, was bitten on the right hand by a fairly large rattlesnake at about 3 P.M. He promptly applied a string tourniquet around his right wrist, and then had to walk more than 3 miles to the nearest habitation. He did not reach the hospital until 7 P.M., when the wound was incised and treatment with antivenin was begun. During the course of the next few days the right arm was very swollen and it remained discolored for 6 days. He was discharged home on the tenth hospital day without any residual damage.

The most important aspect of treatment is the giving of adequate quantities of antivenin (Fig. 6). The vast majority of snake bites in this

country are caused by rattlesnakes, cotton-mouth moccasins, and copperheads. All these snakes are of the same biologic family and their bites can be treated by the same type of antivenin (antivenin crotalidae polyvalent, Wyeth Laboratories, Inc., 1401 Walnut Street, Philadelphia 2, Pa.). The bite of the coral snake is much more rare. This snake is related biologically to the Indian cobra and antivenin is obtainable from the Institute of Butantan, Caixa Postal 65, São Paulo, Brazil. The antivenin can be given locally around the wound intramuscularly and intravenously. The dose ranges between 10 and 50 ml., and depends upon the severity of the bite and the size of the patient. Alcohol should not be given because its depressant actions are synergistic with those of snake venom. Cortisone or ACTH may not reduce the mortality, but sometimes relieves some of the symptoms. Allergic responses to antivenin may be treated with antihistamines, but the patient should first be tested for sensitivity to the serum.

Antivenin will keep in the dry state for many years. Therefore, every doctor who thinks there is even a slight likelihood of his having to treat snake bite should keep a supply of antivenin available. Anti-snake bite kits are available to the public. People who plan to camp, fish or hunt in snake-infested areas should be encouraged to carry these kits with them.

SPIDER BITE[17]

The possibility of being bitten by a spider is almost as frightening to the average person as the possibility of being bitten by a snake. The bites of spiders are not uncommon in many areas but the exact incidence is difficult to ascertain since many persons are bitten without ever finding out exactly what bit them.

Black Widow Bite

The commonest biting spider is the black widow which, characteristically, is found most commonly in outhouses, basements and backyards. This spider likes to live in dried wood piles, brush piles, and the dark corners of barns, garages and houses. Only the female of the species is dangerous. She is jet black and globular in appearance (Fig. 7). On the abdomen there is a red mark shaped like an hourglass.

Two-thirds of the patients with black widow spider bite admitted to this hospital received their bites while asleep in bed. In each of these cases, the spider was killed and identified. There has been at least one other report of a bite due to a different species of spider, sustained while the patient was asleep in bed.

The victim immediately feels a sharp pain at the site of the bite, and in a very short space of time a small wheal with an area of erythema ap-

Fig. 7. Abdominal view of female black widow spider showing "hourglass" marking. (From the Van Riper Collection, University of Colorado.)

pears. The most prominent symptom thereafter is generalized muscle spasm. If the bite is sustained on an extremity, the spasm occurs not only in the limb but also in the chest and the abdominal muscles. The symptoms of abdominal muscular spasm and pain may be extremely confusing. The patient may present to the emergency room of the hospital complaining primarily of abdominal pain; in fact, if the bite was received while he was asleep he may have forgotten about the initial pain at the site of the bite. All the patients admitted to this hospital were found to have rigid but not tender abdomens.

CASE IX. This patient, aged 29 and the wife of a physician, arose one morning in the usual manner, dressed, and began to prepare breakfast. A few minutes later she felt a sharp pain in her right big toe, as though she were treading on a nail. She examined the toe but could find nothing. Within 15 minutes she had very severe muscular cramps of the same leg and began to feel nauseated. The pain in the toe continued and when she tipped out her shoe a large black widow spider emerged, making the diagnosis clear. The muscle cramps increased and became generalized, and nausea and vomiting were constant. Demerol was required for relief of the pain, which was not helped by intravenous calcium gluconate. She was also given cortisone. Severe symptoms persisted for 48 hours, and it was a week before she had completely recovered.

TREATMENT. Death from black widow spider bite is rather uncommon, but the patient may have considerable pain and discomfort in the first 24 to 48 hours. The treatment consists largely in general measures.[18] Morphine or codeine may be given for pain, and calcium gluconate 10

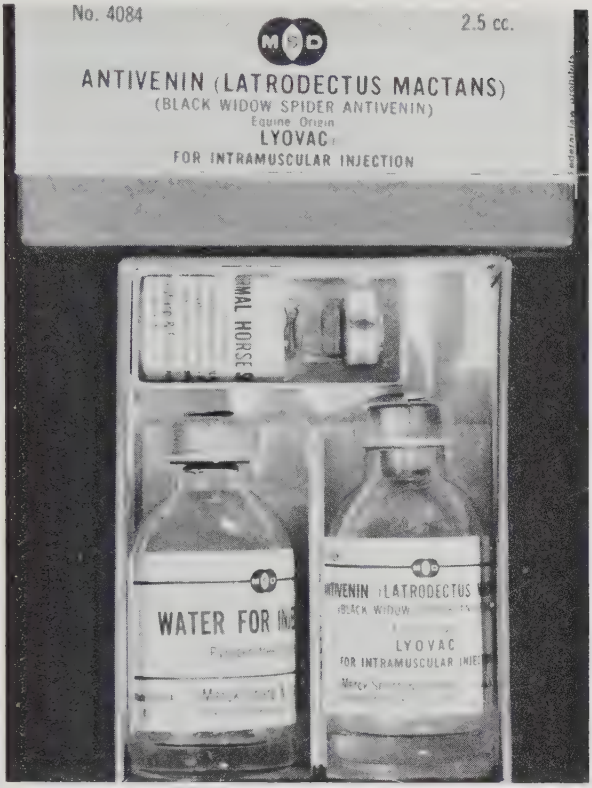

Fig. 8. Contents of anti-black-widow spider antivenin package. "Lyovac" Antivenin is supplied in one "Vacule" vial containing not less than 6000 antivenin units (with preservative: Thimerosal, 1:10,000). Restore by adding the 2.5 cc. of pyrogen-free, sterile Water for Injection in the accompanying vial. A 1 cc. vial of normal horse serum (1:10 dilution) for use as a test and desensitizing material is also included.

ml. of 10 per cent solution intravenously may, in some instances, have a dramatic effect on the muscle spasm. Recently, the use of muscle relaxants such as Tolserol has been suggested.[19] Specific antivenin should be used and is available from Merck, Sharp & Dohme in a dried preparation with separate diluent. It keeps for many years (Fig. 8).

Brown Spider Bite

In recent years, the danger of bites of species other than the black widow spider has been documented. In particular, a syndrome of necrotic arachnidism has been described by Atkins[16] and others from the University of Missouri. The spider responsible is a small undistinguished looking brown spider (Loxosceles reclusus), which is commonly found in the Missouri valley and other areas of the midwest. The bite causes immediate local pain, which may be intense. In the first 24 hours after

the bite occurs, there may be an extensive extravasation of blood into the surrounding area, and a generalized scarlatiniform eruption may appear over the body. The area of the bite becomes deeply ulcerated, and the ulceration may extend down to the muscles. In one case, it took ten weeks for the area to heal. Although clearly documented cases of human bite due to this spider are rare, there is a great deal of circumstantial and presumptive evidence that this syndrome occurs not uncommonly in humans due to the bite of this spider. An exactly similar syndrome has been produced in experimental animals by inducing the spider to bite the animal and observing the subsequent illness. Spiders of this sort have been regularly found in houses of patients who present with this type of syndrome and, therefore, any patient in these geographical areas presenting with obscure painful ulceration, possibly due to insect bite, should be considered to be the victim of brown spider bite. No specific antivenin or treatment is as yet available.

SUMMARY

Bites of animals are common injuries. The morbidity from them and the anguish caused by them is considerable, although fatalities from them are relatively uncommon.

The injuries caused by human, dog, snake and spider bites have been discussed and treatment outlined.

Many of these injuries could be avoided by common-sense practical measures, and prompt treatment also reduces the danger arising from them.

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Acute Tubular Necrosis and Its Management

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The occurrence of oliguria following surgery, transfusion reaction, nephrotoxic agents or trauma represents a serious complication which requires constant attention to metabolic details. When oliguria is discovered, initial efforts should be directed toward determination of its cause. Complete anuria is usually secondary to an obstruction that can be corrected by urological intervention. Shock, if present, must be treated before further evaluation can be accomplished. Extrarenal causes for oliguria such as dehydration, hyponatremia and liver failure must be ruled out. Laboratory tests may aid in establishing the diagnosis of acute tubular necrosis. If it is present, the urinary specific gravity will range between 1.008 and 1.014. Proteinuria is usually a finding. The urinary sodium and chloride concentrations will range from 50 to 90 mEq./L. This is of special diagnostic significance in the presence of a low serum sodium concentration and is in contrast to the reduced urinary sodium concentration of hyponatremia. The urinary urea nitrogen will fall to 300 to 400 mg./100 ml. and the urinary osmolality to 300 to 350 mOsm./L. Finally, a phenolsulfonphthalein excretion test will show 0 to less than 5 per cent dye excretion. These findings together with a distinctive clinical sequence are the most useful criteria for establishing the diagnosis.

Once a diagnosis of acute tubular necrosis has been established, the physician should set in motion carefully planned procedures for care of the patient and for metabolic evaluation. From 1953, when hemodialysis was first initiated at this hospital, until the present, the renal service has treated 110 patients with acute tubular necrosis requiring hemodialysis. Observations made during treatment of this group will form the basis of this report.

METHODS AND TECHNIQUES

Since a majority of these patients were transferred from other hospitals, the initial phases of their treatment often were not under our control.

All were treated in the medical intensive therapy unit during the acute phase of renal failure. The majority had indwelling catheters and intakes and outputs were recorded every six hours. The patients were weighed once a day.

Dialyses were performed in a special room, employing the conventional Kolff twin coil technique.[10, 11, 12] For dialysis, tapered plastic catheters were inserted in the radial artery and a large forearm vein. In patients with systolic blood pressure below 110 mm. Hg, a catheter inserted into the vena cava was substituted for the radial artery catheter. Prior to 1959 one mg./kg. of heparin was given, and doses were repeated as needed to maintain a clotting time above 20 minutes (Lee-White). Since 1959 regional heparinization has been used routinely.[8] The coil was primed with two bottles of bank blood and the patient's blood circulated through the coil at the rate of approximately 300 ml./min. Duration of dialysis ranged from four to eight hours, with an average of approximately five and one-half hours. Composition of the bath solution was varied as needed to correct solute concentrations in the patient's body fluids.[10]

Several techniques using special equipment have proved of value in following these patients. A Jarvis stretcher scale was used to obtain daily weights on all patients. A Howe platform scale* accurate to one-quarter pound was modified to support a stretcher on which the patient rests throughout dialysis.[9] By periodic weighing, fluid balance could be rigidly controlled during dialysis. The accuracy of the various balances was checked frequently by test weights. A temperature-compensated hand refractometer† was used to obtain accurate urinary specific gravities on volumes as small as 1 ml. and the percentage of total solids using only a few drops of serum. The measurement of osmolality by the freezing point method was used in evaluating the nature of increases in total body solute. The periodic measurement of salivary flow in these patients was of use in focusing attention on mouth care and in helping to evaluate the fluid requirements.[7]

MANAGEMENT AND RESULTS

The age distribution in this group was quite wide. There were no patients under ten years of age. Approximately 50 per cent of the patients were over 50 years of age and almost a third above the age of 70.

For purposes of analysis the patients were grouped according to the circumstances leading to acute oliguria. These categories included post-surgical problems, post-traumatic problems, nephrotoxic agents (carbon

* Howe Weightograph 9808 (300 pounds capacity).
† TS meter manufactured by American Optical Company.

tetrachloride, bichloride of mercury, etc.), acute septicemia, and abortion and pregnancy problems. The largest number of patients fell in the postsurgical group. In the postsurgical and post-traumatic groups further subdivision was made into those having associated identifiable shock, those with transfusion reaction, and those with severe infection and associated septicemia. The results of this grouping are shown in Table 1.

Table 1. The Clinical Events Preceding the Development of Acute Tubular Necrosis (110 Patients)

CATEGORY	PER CENT
Postsurgery	48.5
Post-trauma	12.5
Nephrotoxic agents	11.5
Septicemia	9.5
Abortion and pregnancy	10.5
Miscellaneous	7.5
Postsurgical and Post-trauma	
Transfusion reaction	12.5
Shock verified	75.0

These factors are generally accepted as contributory to the development of acute tubular necrosis. Evidence is still lacking as to the precise pathogenesis of acute tubular necrosis. Perhaps metabolic factors contribute. For example, in our series 95 per cent of the patients had a reduction of the plasma cholinesterase below the normal range, whereas a group of postsurgical patients who did not develop acute tubular necrosis showed no deviation from the normal. Eight patients in this group had no shock during surgery or immediately after accidents, but developed shock 12 to 24 hours later. Often the shock was undetected for an indeterminate period of time. This emphasizes the need for careful observation of the patient during the first 24 hours after surgery or trauma.

Specific Therapy for Shock, Transfusion Reaction or Infection

The first phase of treatment in a patient with acute oliguria involves specific therapy for shock, transfusion reaction or infection. In the case of shock, prompt evaluation of fluid and blood volume needs is essential so that proper replacement therapy can be instituted. Laboratory aids useful for this evaluation include measurement of the blood volume, determination of hematocrit and total serum solids, and the measurement of central venous pressure as described elsewhere in this symposium by Dr. Wilson. Pressor agents should be considered as a temporary measure to be used only until proper fluid or blood volume adjustments

can be achieved. A high urinary specific gravity at this time is considered indicative of an oliguria secondary to fluid and electrolyte deficiencies.

When a transfusion reaction occurs, tests should be made for the presence of hematuria or hemoglobinuria. Their presence dictates the establishment of a prompt diuresis either with water load or by infusion of mannitol solutions (20 per cent) to induce an osmotic diuresis.[2] The initial intravenous dose of mannitol should be limited to 100 grams. Only when there is a prompt diuresis should additional amounts be given. Alkalinization of the urine can be achieved by administration of sodium bicarbonate or sodium lactate.

Infection should be treated with appropriate antibiotic therapy, the organism being identified and sensitivities determined as rapidly as possible. If oliguria persists, the maintenance dosage of the antibiotics should be reduced to 50 to 75 per cent of the dose used for non-oliguric patients.

It is then necessary to establish the diagnosis of acute tubular necrosis using the criteria previously cited. Some unusual causes for acute oliguria observed by this unit include acute shock, genitourinary manipulations, multiple myeloma, acute lymphatic leukemia, emboli of the renal arteries, renal vein thrombosis, operative section of the ureter, obstruction of the ureters by tumor, hyponatremia, and acute intracranial lesions.

Conservative Management of the Acute Oliguric Phase

Once the diagnosis of acute tubular necrosis has been established, the second phase of treatment is instituted—conservative management of the acute oliguric phase.[3, 4, 15, 17]

CONTROL OF FLUID BALANCE AND THIRST. The most important aspect of treatment in this phase is proper control of the fluid balance. Body weight is measured daily and is kept constant throughout the oliguric period. This is in contrast to a weight reduction of 0.3 kg. per day suggested by Merrill.[15] For initial calculations we usually allow about 600 ml. per day for insensible water loss in winter and up to 1000 ml. in summer. The output of all types as through gastric suction, urine and wound drainage is measured every six hours. An order is then written to replace the measured output plus 150 ml. (based on a calculated insensible loss of 600 ml./day) every six hours. With such an order, there is no possibility of the patient's being over or under his actual fluid requirements for more than six hours.

These patients complain of severe thirst and have marked dryness of the mouth even though they may have associated edema.[7] Actual measurements of salivary flow indicate a significant reduction during this period (Table 2). This dryness undoubtedly contributes to the complaint of intense thirst. It has been suggested that decreased salivary

Table 2. Comparison of Salivary Flows (Mouth-Breathing Technique) in Patients with Renal Failure

Average Flow (ml./5 min.)

		RESTRICTED FLUIDS		UNRESTRICTED FLUIDS	
	NUMBER	1	2	3	4
Acute failure	21	0.23	0.23	3.1	3.8
Chronic failure	20	0.32	0.58	2.1	2.5
Renal disease without failure	16	—	—	2.4	3.2

Table 3. Electrolytes in Saliva

TYPE	Vol. (ml./5 min.)	K (mEq./L.)	Na (mEq./L.)	Cl (mEq./L.)
Mouth Breathing				
Restricted fluid	0.85	45.0	20.5	39.8
Convalescent	2.92	26.5	13.3	27.5
Non-failure	1.86	27.3	8.1	21.2
Gum Chewing				
Restricted fluid	6.67	33.6	25.8	27.6
Convalescent	8.10	23.5	26.2	29.7
Non-failure	7.57	22.9	5.0	18.5

flow is caused by a reduction in circulating blood volume to the salivary glands and corresponds to the reduction in salivary flow described in dehydration.[1] However, there may be inherent changes in the salivary glands in uremia since the electrolyte concentrations of the saliva are quite different from the normal as shown in Table 3.

Most physicians are aware of the complications that may be associated with fluid overloading, but few realize that serious complications may also result from too vigorous fluid restriction. One is shock due to hyponatremia and dehydration. We lost two patients who suffered obstruction of the trachea or one of the main bronchial passages, due to accumulation of layers of inspissated mucus to the point of obstruction. Subsequently, tracheotomies were done on two other patients because of similar obstruction. Another complication of dehydration is parotitis. When possible, the patient is asked to chew gum three times a day, since stimulation of salivary flow may tend to prevent parotitis. Mouth care is extremely important in these patients. Oral ulcerations are quite common. A mixture of equal amounts of glycerin and lemon juice may be helpful for mouth care. Frequent use of popsicles has proved valuable in relieving the patient's oral discomfort.

It is well documented that gastric factors play an important role in the satisfaction of thirst.[16] The same amount of fluid given orally will usually relieve thirst much more effectively than when given intravenously. Therefore, the patients are encouraged to take all fluids by mouth. Oral food intake is encouraged as long as possible. The diet prescribed contains 20 grams of protein, 1200 calories, with potassium and sodium content restricted as dictated by the blood chemistries. While such a diet is not very attractive it does more nearly satisfy the patient's desire for food than do butter balls and borscht soup. We also use a "renal cookie" which is permitted ad lib. (Each cookie contains 3.3 gm. carbohydrate, 0.6 gm. protein, 3.0 gm. fat and no sodium or potassium.) When oral feedings are not possible, a 500 ml. bottle of 50 per cent glucose (containing insulin 1 unit per 4 grams of glucose) is given intravenously over a four to six hour period each day.

BLOOD TESTS AS GUIDES IN TREATMENT. What blood determinations are essential for following these patients? After establishment of the diagnosis of acute tubular necrosis, serum sodium, potassium, chloride, CO_2 combining power, creatinine and blood urea nitrogen are studied. Particular attention is given to the "R" fraction (undetermined anion fraction), which is calculated by subtracting the sum of the CO_2 combining power and chloride concentration from the sodium concentration. Normally, the "R" fraction is 12 mEq./L. In our patients this figure averaged 40 mEq./L. prior to dialysis. Of the various tests, those for blood urea nitrogen, CO_2 combining power, "R" fraction and serum potassium have the most influence in the decision to perform dialysis. These five determinations are therefore requested daily until a definite pattern of change is established for each patient. This is particularly important in trauma cases, in which values may increase quite rapidly, in contrast to the case of uncomplicated carbon tetrachloride intoxication, in which changes occur quite slowly. Once the rate of increase is established, determinations on alternate days may be sufficient.

CONTROL OF HYPERKALEMIA. During conservative therapy, hyperkalemia must be controlled. It is not a problem in approximately 59 per cent of the cases, as indicated by the potassium levels obtained at the time of initial dialysis (Table 4). However, after trauma serum potassium concentration rises rapidly, presumably due to absorption from injured tissue. When the serum potassium concentration approaches 6.2 mEq./L., dialysis should be instituted in the next 24 hours unless a decline occurs as a result of specific therapy. There are three accepted methods for controlling hyperkalemia. The first is the administration of glucose and insulin, which tends to lower serum potassium by causing a shift intracellularly. The second is the administration of sodium bicarbonate, which also causes potassium to shift intracellularly. This method is particularly applicable when the patient has a marked acidosis.

Table 4. The Spread of the Initial Predialysis Serum Potassium Values in 106 Patients with Acute Tubular Necrosis

SERUM POTASSIUM (mEq./L.)	NUMBER	PER CENT
3.0 to 4.0	29	27.0
4.0 to 5.0	34	32.0
5.0 to 5.5	13	12.5
5.5 to 6.0	11	10.5
6.0 to 6.5	9	8.5
6.5 to 7.0	9	8.5
Above 7.0	1	1.0

In some patients the serum potassium concentration has dropped as much as 2 mEq./L. following the intravenous administration of 120 to 160 mEq. of sodium bicarbonate. The third method is the administration of cation exchange resins. These are usually given orally as sodium resins in doses of 15 grams four times a day. Each gram of resin will remove between 1 to 2 mEq. of potassium and add to the body fluids 2 to 3 mEq. of sodium. The added sodium also has an effect similar to sodium bicarbonate, producing an intracellular shift of potassium.

Indications for Hemodialysis. The principal signs of hyperkalemia are changes in the electrocardiogram, ascending quadriplegia, and paralytic ileus. The electrocardiographic changes which are considered an indication for immediate dialysis are spreading of the QRS segment, prolongation of the P-R interval, fibrillation, and bundle branch block type changes in the QRS complex. However, serious hyperkalemia may exist in the absence of electrocardiographic abnormalities. For example, one hyperkalemic patient with ascending quadriplegia which disappeared completely during the first two hours of dialysis had no electrocardiographic changes. Likewise, several patients with paralytic ileus of the bowel had sudden return of bowel sounds during the first two hours of dialysis and showed no significant electrocardiographic changes.

In patients with acute tubular necrosis following trauma the blood urea nitrogen concentration often shows a daily rise of 30 to 50 mg. per 100 ml. In contrast, during oliguria secondary to carbon tetrachloride poisoning the rise may be only 10 mg. per 100 ml. per day. In our experience, a blood urea nitrogen between 125 and 150 mg. per 100 ml. represents the level at which serious complications may occur. Thus a blood urea nitrogen between 125 and 150 mg. per 100 ml. is considered by us as indication for dialysis.

The most feared complication in trauma cases is the development of convulsions. Ordinarily the onset of convulsions cannot be predicted clinically, but in several instances there was a prodromal period during

which the patient complained of difficulty in reading, diplopia, and other signs of central nervous system dysfunction.

A decrease in the CO_2 combining power below 12 mEq./L. is usually associated with increasing symptomatology. Therefore a CO_2 combining power below 12 or an increasing "R" fraction to or in excess of 30 mEq./L. is considered an indication for dialysis. Often the patient will show definite clinical improvement after intravenous injection of sodium bicarbonate. It is probable that the symptoms are secondary to a serum pH change, but there is insufficient data to determine the usefulness of the blood pH measurement in evaluating progressive uremia.

The determination of serum osmolality may be useful in indicating an unusual metabolic change which can be corrected by dialysis. Normally, the difference between the measured milliosmols and the value calculated from the sodium concentration, the blood urea nitrogen and the glucose concentration should be 7 to 10 mOsm./L.[5, 17] In some uremic patients this difference may be 50 to 90 mOsm./L. and in our experience tends to be associated with greater toxicity.

An excessive fluid load, particularly when associated with cardiac failure, can also be considered a definite indication for dialysis. During a six to seven hour hemodialysis, as much as 18 pounds of fluid have been removed. The Kolff twin coil unit is particularly adapted for fluid removal, since filtration pressures of over 200 mm. of mercury can be maintained in the dialysis unit.[9]

The clinical signs and symptoms of uremia are still among the best indications for dialysis. These include progressive nausea and vomiting, mental depression and disorientation, convulsions, muscular twitching, etc. The patient who can continue taking food and fluids by mouth and who remains alert and mentally clear appears to tolerate uremia well and have a shorter hospital stay. In Table 5 can be seen one of the best arguments for early and frequent dialysis, a policy recently advocated by Teschan and others.[20, 22] Our patient group is divided into those treated from 1953 through 1958 and those treated from 1959 to the present. In the first series the mortality was 53 per cent, a figure com-

Table 5. Acute Tubular Necrosis Requiring Hemodialysis

	1953–1958	1959–PRESENT
Number of patients	58	52
Mortality	53%	22%
Death during first 48 hours	26%	40%
Death during diuretic phase	33%	9%
Number of dialyses (average)	1.4	2.0
Blood urea nitrogen over 200 mg./100 ml.	50%	22%

parable to that reported by Doyle for several different series.[3] After 1958 the mortality was 22 per cent. This improvement can be attributed to several factors including: (1) early and more frequent dialyses, (2) improved dialysis technique, and (3) earlier transfer of patients to the dialysis center. Table 5 shows that, prior to 1959, 50 per cent of the patients had a blood urea nitrogen exceeding 200 mg./100 ml. at the time of the first dialysis, whereas after that date only 22 per cent fell in this category. In the first group there was an average of 1.4 dialyses per patient; in the second group 2 dialyses per patient. In the early experience, 33 per cent of the deaths occurred in the diuretic phase, whereas only 9 per cent occurred in this phase after 1958. Prior to 1958 dialysis was rarely performed in the diuretic phase. Now dialysis is carried out immediately during the diuretic phase if there are any clinical signs of toxicity. There are still a substantial number of deaths in the first 48 hours. If the concept that early and frequent hemodialysis offers the best prospect of reducing mortality in acute tubular necrosis is correct, patients should be transferred to dialysis centers shortly after the diagnosis of acute tubular necrosis is established.

In trauma cases, where the average patient requires four dialyses during the oliguric phase, the problem has been simplified by employing an arteriovenous shunt using permanent Teflon catheters inserted in the radial artery and forearm vein as described by Scribner et al.[18, 22] The catheters are specially shaped for each patient, and can be readily hooked to the dialysis unit. This same shunt could also be used for resin columns when they are developed to the point of being practical and effective for potassium removal.

There are virtually no contraindications to hemodialysis. Shorter dialyses tend to eliminate some of the hypotensive and hypertensive changes which may occur during six-hour dialyses. Regional heparinization is now used routinely; therefore bleeding is not considered a contraindication to dialysis. In many patients the clotting time may be appreciably shortened by dialysis due apparently to removal of circulating antithrombin and antiplatelet factors.[21] Rapid removal of potassium in a digitalized patient may produce arrhythmia, but this can be controlled by careful regulation of the potassium concentration in the bath and by shorter and more frequent dialyses. Coronary occlusion is still considered a contraindication, but such patients have been dialyzed successfully.

Treatment of Infection. Infection is the most serious complication during the oliguric phase. Most uremic patients are hypothermic; therefore the temperature curve is not a good index of the onset of infection. A spiking temperature curve, however, even though the highest temperature does not exceed 37.8° C., may indicate infection. The white blood cell count is actually a much better guide to the presence of infection.

White counts of 40,000 cells per cubic millimeter have been observed when the temperature was still within the normal range. The Teflon shunt may become infected and should be inspected frequently. A careful watch for infection and prompt treatment are mandatory in this group.

Because of the exaggerated metabolic responses in uremia, *drug therapy* should be rigidly restricted and given only when there is a definite indication.[13] For example, nitrofurantoin (Furadantin) can produce an acidosis which is always more severe and prolonged in the uremic patient.[6] Tetracycline has a catabolic effect[19] which may raise the blood urea nitrogen and serum potassium concentrations significantly. It is likely that other antibiotics may produce significant metabolic effects in uremic patients. For most antibiotic therapy the usual initial dose is given, and the maintenance dose is reduced to between 50 and 75 per cent of that given a patient with good renal function.

Management of the Diuretic Phase

The third phase of treatment starts with the onset of diuresis. As urinary volume increases, the blood urea nitrogen will continue to rise until the diuresis reaches approximately 2000 ml. per day. In these patients the urinary urea nitrogen concentration ranges between 300 and 350 mg./100 ml. On the low protein diet prescribed, the total urea nitrogen excretion ranges from 4 to 5 grams per day. With these figures one can predict at what urinary volume the blood urea nitrogen will start to drop. During the phase of rising blood urea nitrogen, progressive clinical signs of uremia are considered an indication for dialysis. In the diuretic phase the urinary sodium concentration is fixed between 50 and 90 mEq./L., with an average concentration of 70 mEq./L. This requires salt replacement of about 500 ml. of 0.9 per cent saline for each liter of urine excreted. We always measure urinary sodium and potassium concentrations during the diuretic phase, calculate the daily loss by multiplying by the 24-hour urine volume, and replace mEq. for mEq. This will eliminate hyponatremia as a complication of the diuretic phase. It is our policy to replace the urine volume by adequate fluids up to excretion rates of 5 to 7 liters. With this type of diuresis, the blood urea nitrogen and other metabolic changes return to normal more rapidly. This policy has little danger with proper electrolyte and fluid replacement.

Complications Arising During Treatment

Table 6 shows the acute clinical complications recorded on the patients' charts and Table 7 shows the complications found at autopsy. Two patients in this series died from acute cholecystitis, but later two other patients with this complication recovered following appropriate surgery. Acute pancreatitis is a commonly occurring complication and was found in 26 per cent of the autopsied patients. It has also been reported as a

Table 6. Incidence of Common Complications Observed During Treatment of 110 Patients with Acute Tubular Necrosis

TYPE OF COMPLICATION	NUMBER
Septicemia	14
Genitourinary tract infection	12
Pneumonia	9
Cardiac arrhythmias	9
Acute pancreatitis	7
Acute parotitis	7
Respiratory tract obstruction	7
Wound infection	6
Gastrointestinal tract bleeding	6
Peritonitis	6
Pelvic infections	7
Acute cholecystitis	4
Myocardial infarction	3
Anesthetic death	3
Evisceration of wound	3

Table 7. Pertinent Autopsy Findings in Addition to Acute Tubular Necrosis

	PERCENTAGE OF CASES
Pancreatitis	26
Cardiac enlargement	26
Septicemia	16
Liver necrosis	13
Pneumonia	13
Peritonitis	8

contributory cause of acute tubular necrosis.[14] We have been concerned as to whether pancreatic secretions are reduced to the same extent as salivary secretions and whether such reductions may contribute to the high incidence of pancreatitis. Acute parotitis has occurred in a significant number of patients. Peritonitis, septicemia and pneumonia are common infectious causes of death. Cardiac failure represents another serious complication. The above data are useful because they tend to focus the physician's attention on specific areas which must be watched carefully.

Complications of Hemodialysis

Plasma hemoglobin determinations have been made before and at the end of dialysis in approximately 500 instances. Significant changes occurred in less than 2 per cent. Blood cultures were taken before and after dialysis in all patients and in only two instances were pathogenic organisms present at the end of dialysis which were not found at its beginning. In two patients suffering from transfusion reaction, blood for

priming the coil unit could not be obtained because of incompatibilities. In these cases, the coil was primed with Albumisol.* When the hematocrit was low only one coil was used, thus reducing the required priming volume by one-half, and the dialysis time was doubled. Hypotension and hypertension have been observed during dialysis, usually after the second or third hour. The hypotension can usually be corrected by increasing the blood and fluid volume. Hypertension has been treated with various drugs, the most successful of which has been intravenous reserpine. Headache may be troublesome.

Because of the rigid controls and careful observation, serious complications rarely occur during hemodialysis. When death occurred, it was usually during the first five to six hours after completion of dialysis, and in several instances was due to an inadequate airway. Patients should be observed carefully during the first 12 hours after dialysis. In acute tubular necrosis in the diuretic phase, there is usually no post-dialysis oliguria.

RECAPITULATION

Discovery of acute oliguria following trauma or surgery demands the physician's immediate attention to establish the cause of the decreased urine flow. After establishment of the diagnosis of acute tubular necrosis, metabolic changes must be carefully observed and appropriately controlled to prevent death. Unfortunately, some physicians look upon acute oliguria as a problem to be managed only by conservative means. Dialysis provides another tool for use in the treatment of this condition. Evidence is accumulating that early and frequent dialysis may prevent many of the complications formerly observed with conservative management. By using this technique it is possible to keep the patient alert, eating, and taking fluids by mouth during the oliguric phase. The hazards of dialysis are small, and can be further reduced by application of the technique before the patient reaches a preterminal or moribund state. When urine flow increases during the diuretic phase, the condition is still critical, and dialysis should be employed whenever the previously listed indications are present. For example, in one instance hyperkalemia (8.2 mEq./L.) with extreme cardiac dysfunction (sine wave EKG) presented in a patient with a urine volume of 2000 ml. per day. Current results indicate that frequent and early use of dialysis plus good conservative management is attended by recovery in a high percentage of patients with acute tubular necrosis. Further reductions in mortality will follow upon realization that surgical conditions can be managed by standard techniques and be well tolerated if metabolic changes are minimized by appropriate dialysis.

* Five per cent normal human serum albumin (Merck, Sharp and Dohme.)

It seems logical that patients with acute tubular necrosis should be managed in centers devoted to this problem. Adequate staffing and the availability of numerous laboratory aids necessary for proper management are minimal requisites. With experience, many of the complications and problems that were encountered earlier can be avoided. In our most recent series of cases, 40 per cent of the deaths occurred during the first 48 hours after admission. It is believed that earlier transfer of patients to a dialysis center will result in further reduction of mortality in patients with acute tubular necrosis. Probably this is best done as soon as the diagnosis is established.

Traumatized patients have particularly serious metabolic problems after development of acute tubular necrosis as compared, for example, with patients with similar renal lesions after ingestion of carbon tetrachloride. As judged by the rate of rise of the blood urea nitrogen, metabolic changes are probably accelerated threefold or fourfold. It is believed that the absorption of damaged tissue contributes significantly to this process and increases the hazard from hyperkalemia. The dangers of infection are multiplied and the superimposed effects of antibiotics lead to further difficulties. The best results have been obtained when hemodialysis was used every two to three days during the early phases of oliguria.

SUMMARY

Acute tubular necrosis is an infrequent but very serious complication of trauma and occasionally of surgical operations. Experience in the management of this condition over the past eight years has been analyzed and recorded, and current methods of treatment set forth. These include the specific details of conservative measures that can be applied and the use of renal dialysis, which was used in 110 patients. During the earlier phase of experience with these techniques, extending from 1953 through 1958, the salvage rate was 47 per cent. Since 1958, the recovery rate has risen to 78 per cent. This improvement is attributable to earlier referral, improved conservative management, early and more frequent dialysis, and better dialyzing techniques.

Current indications for dialysis include progressive hyperkalemia, decline in CO_2 combining power to 12 mEq./L. or below, elevation of blood urea nitrogen to the range of 125 to 150 mg./100 ml., excessive fluid retention, and clinical signs of progressive uremia.

Nonrenal complications were the most frequent cause of death. These, along with the primary pathologic conditions, were the main deterrents to recovery.

Further improvement in the results will depend not only upon earlier diagnosis and treatment of acute renal failure, but also upon recognition

and control of complications by whatever means are applicable, including surgery when necessary.

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